THE SIGNIFICANCE OF DISORDERED MUSCLE ACTIVITY IN THE PERPETUATION AND TREATMENT OF LOW BACK PAIN, WITH PARTICULAR REFERENCE TO THE EFFECT OF MANIPULATION.

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PREFACE

According to Burke (1964) Sir James Paget made the following statement in an article in the British Medical Journal in 1867:

"Learn to imitate what is good and avoid what is bad in the practice of bone-setters. Fas est hoste doceri (It is right to be taught by the enemy). "

The conviction is held that manipulation has a valid role in the treatment of some spinal pain syndromes. A selected group of patients with predominantly one sided low back pain are studied in detail. Using simple objective measurements a statistically significant change after manipulation is demonstrated. The significance of this change and its relevance to altered motor nerve excitability is discussed.

Burke, G. L. (1964). Backache From Occiput to Coccyx. p. 15. Vancouver : Macdonald.

SYNOPSIS

This thesis is made up of five separate parts, each part constitutes a progressive step in the whole research project.

1. Describes the passive hamstring stretch test which is unknown to most allopathic physicians. The accuracy of the test is researched, using independant trained examiners. Its value in both the clinical and research setting is discussed.

2. Explores the interrelationship between this test and changes in tension as the leg is raised. The relaxed straight leg is raised by a pulley system which incorporates a linear tension gauge. There is a close correlation between a marked increase in tension and the clinical end point of the passive hamstring stretch test. The value of the tension gauge measurements as an objective research tool is discussed.

3. Consists of a pilot study of a patient with one sided low back pain associated with unilateral reduction in the passive hamstring stretch test on the painful side. Both clinical and tension gauge measurements are taken before and after manipulation. A marked change takes place in both measurements

after manipulation of the painful side. Follow up checks of both measurements show that this change relates to the patient's recovery.

4. This is a controlled trial of the effect of a specific manipulative technique on ten controls and ten patients with predominantly unilateral symptoms and restricted passive hamstring stretch on the painful side. The tension gauge measurements show a highly significant and statistically valid change in the patients as compared to the controls. Follow up studies show that further tension measurements reflect improvement in the patients' symptoms.

5. Consists of a study of the effect on the tension measurements of fixing the ankle joint at varying degrees of dorsiflexion. Increased dorsiflexion has a marked effect on these measurements.

It is argued that the sum total of these findings point to a segmental increase in the excitability of the alpha motor neurones on the painful side that has the potential of becoming self perpetuating, and that this is probably mediated through increased gamma motor nerve activity. It is also argued that manipulation and other modalities of treatment have their effect on this increased gamma outflow by stimulating high threshold large fibred inhibitory proprioceptors.

Further possible avenues of neurophysiological research are discussed.

SURVEY OF PREVIOUS WORK

In the management of spinal pain syndromes there are advocates of many differing regimes of treatment. Each advocate has his own theory, some of which seem rational and plausible whilst others are obvious systematised delusions. There are undoubted cures and undoubted failures, but none have been proved to be superior to any other, nor more effective than no treatment at all.

Clinical research into treatment of spinal pain is hampered by the lack of measurable objective signs and our lack of understanding of pain and possible pain provoking mechanisms. It has also been neglected because of the relative benign nature of most instances of low back pain and their tendency to spontaneous recovery. Probably only one or two in every thousand attacks of back pain are severe and prolonged enough to warrant hospitalised investigation and surgery. This has tended to concentrate research on serious disc pathology, but the disc is only one part of a very complicated joint complex with its supporting structures.

Undoubtedly the disc is unduly prone to premature breakdown and ageing and it seems reasonable to suppose that this will lead to altered function in the whole joint complex and supporting structures, making them more vulnerable to strain and possible pain provoking situations, but it seems unlikely that it is the prime source of pain

in most attacks of backache.

If not the disc then what is the source of this common complaint in the majority of cases? Nachemson (1976) takes a critical look at the conservative management of back pain. He makes the point that back troubles start in the 20s, reach their peak between 40 and 50 and diminish thereafter. This coincides with the onset and the peak of degenerative disc disease, whereas degenerative changes in the facet joints, the ligaments, and muscles progress steadily with increasing age. This implies that progressive degenerative changes are synonymous with increasing discomfort. This is patently not true. What seems more obvious is that the incidence of problems occur when enough change has taken place at a time when potentially hazardous activity is still taking place. Either that or the more elderly have realised the futility of complaining.

Because the disc does not seem to be the complete answer to the majority of attacks of back pain, attention has been drawn to other parts of the joint complex, in particular, the facet joints. Lewin (1965) showed that these joints behave like synovial joints elsewhere in the body. Evidence of degeneration in the facet joints usually followed degenerative changes in the discs. It has been suggested that these joints may get impacted or jammed, like a partly shut drawer (Cailliet 1968).

Crisp (1960) suggested that the synovial membrane may get entrapped. Meniscoids have been demonstrated in the ventral and dorsal margins of the facet joints in the cervical and lumbar spines (Kos & Wolf, 1971). These are richly innervated with nociceptive nerve endings, and it has been suggested that these meniscoids may become trapped. It has also been suggested that entrapment of a loose cartilagenous body may be responsible for some attacks of acute backache. Farfan (1973) has found evidence of previous facet joint sprains in his biopsy studies.

It would seem unlikely that these hypothetical causes could account for more than a small minority of attacks of acute backache. Previous studies have shown that anything from 44 to 88% of patients do not remember putting their backs to any undue strain prior to the onset of the pain (Ghormley 1951, Hirsch 1956, Dillane et al 1966, Fisk 1977). In a previous retrospective study (Fisk 1977) an attempt was made to classify attacks of backache according to the precipitating strain (severe, mild, protracted, nil) and the nature of the onset of the pain (sudden or gradual). All the patients in this study were treated by a specific manipulation technique, at least, in the initial stages. The idea behind this was to try and identify low back pain syndromes that recovered quickly whilst being treated, that is, to try and find patterns of low back pain that could conceivably be benefited by this type of treatment.

The specific manipulative technique used is known as a high velocity, short amplitude thrust. The thrust is applied as near to right angles to the plane of the facet joints as is anatomically possible. The facet joints are positioned so that the two surfaces are in full apposition. This is described in detail elsewhere (Fisk 1977A, see appendix). This high velocity thrust produces a clicking or clonking sound that is similar to the sound produced by knuckle cracking. Unsworth et al (1971) showed that this sound is produced by the bursting of tiny bubbles of gas which are created by vapourisation of the fluid on the joint surfaces. This results from the negative pressure caused by the traction on the joint. It takes up to 20 minutes for this gas to be reabsorbed and the sound cannot be reproduced during this time. That the same thing happens in the facet joints of the spine is implied by studying the planes of the joints and the angle of thrust. The joint is in the neutral position and the sound could not conceivably come from anywhere else. It is impossible to reproduce the sound for at least 20 minutes and it is easier to produce the sound in normal spines than in those with problems, due to the difficulty created by associated muscle spasm.

This retrospective study failed to identify a particular group of patients who recovered quickly. The overall rate of recovery was approximately the same for all the groups. Some of the patients in all of the groups recovered dramatically after being manipulated, and

it seemed likely that although the manipulation involved the gapping of the facet joint the improvement was not necessarily associated with an effect upon the facet joints themselves.

There is now no doubt that most tissues in the body are equipped with a specific system of nerve endings that are particularly sensitive to tissue dysfunction, whether this be chemical or mechanical. These have been categorised as nociceptive receptors. Wyke (1970) showed that in the spine this system is represented by plexiform and freely ending arrangements of unmyelinated and small myelinated nerve fibres that are distributed throughout the skin and subcutaneous tissues, adipose tissue, fascia and ligaments, periostium, dura mater, the adventitia of blood vessels, and the fibrous capsules of the facet and sacro-iliac joints, but not in the articular cartilages or synovial tissue of any of these joints (Wyke 1972).

The small diameter (up to 5 μ) determine their slow conductivity, sensitivity to blockade with local anaesthetic agents and their low sensitivity to direct stimulation of the peripheral nerve branches containing them (Wyke 1970). In normal circumstances these nociceptors are relatively inactive. They may be depolarised by mechanical distortion of their endings or by chemical irritation by substances that are released from traumatised, inflamed, necrotic or ischaemic tissues

(Wyke 1972). They are non adaptive, that is, they keep on firing until the noxious stimulus stops (Wyke 1972). In the low back their cell bodies are located in the dorsal root ganglia of the lumbar and sacral nerves and reach there by three main pathways:

1) Via the cutaneous, muscular and articular branches of the posterior primary rami of the segmentally related spinal nerves (Pedersen et al 1956).

2) Via the recurrent (sinuvertebral) nerves (Pedersen et al 1956).

3) Via branches of the plexus of nerve fibres surrounding the paravertebral system of veins (Wyke 1970).

It is of importance to know that the nerve supply to the spinal joint complex in the lumbar (and cervical) spine has a dense system of ascending and descending branches. Thus, each spinal level receives nerve endings from more than one segmental level. Furthermore, they cross the mid line (Pedersen et al 1956, Edgar et al 1966).

The spinal joint complex may be likened to a three-legged stool, one leg being the disc and the other two being the facet joints. It is impossible to move one leg without involving movement in the other two. In the absence of evidence of interference in nerve conduction in one of the spinal nerve roots, due, for example, to pressure from a large disc protrusion, it thus becomes obvious that the exact localisation and cause (mechanical

or inflammatory) of the painful back is mainly guesswork and the diagnosis made depends on the individual bias of the examining doctor.

Kellgren (1939) attempted to deliniate the segmental distribution of nociceptors in the lumbar spine by injecting hypertonic saline into varying levels of the paravertebral structures, and by mapping out the areas where the subjects experienced pain. These experiments have been repeated by others (Feinstein et al 1954, Hockaday et al 1966, and others). Because of the diffuse distribution of the nociceptor system and because of the widespread intersegmental linkage such attempts to deliniate a segmental nociceptor innervation in the lumbar spine are obviously fallacious. But some important observations did arise from these experiments:

It was noted that the areas of pain varied from individual to individual on stimulation of the same segmental level, but on further stimulation the areas remained constant for each individual. If the subject had suffered previous backache the areas of pain were channelled to the same distribution as the previously experienced pain. The pain was often experienced at a considerable distance from the site of the injection. This has been described as the sclerotomal pattern of pain referral. This referred pain was also often associated with tender focal areas of muscle spasm and altered skin

sensitivity (hyperalgesia). Furthermore, injections of these areas of muscle spasm with local anaesthetics often abolished the pain caused by the original stimulus.

When examining painful necks and backs a common finding is the so-called myofascial trigger points of Travell (Travell et al 1952). The more one looks for them the commoner they become. Controversy exists regarding their nature and treatment. Cyriax (1962) states that they are areas of referred tenderness due to dural nociceptor stimulation and that manipulation of the displaced fragment of disc leads to their disappearance. Bourdillon (1973) believes that they are secondary areas of muscle spasm or fibrositis and failure to treat them as well as manipulating the spine is a common reason for the failure of manipulation of the primary spinal joint lesion. He recommends their treatment by heat, deep friction, or possibly injections of local anaesthetic. Stoddard (1969) states that it is a waste of time treating these areas of muscle tenderness by heat and massage without also dealing with the primary problem in the spinal joint, by manipulation.

On the other hand there are those who believe that deficient muscle activity and trigger points are one of the main causes of back and neck pain (Travell et al 1952, Kraus, 1967, and others). Berges (1973) reviews these co-called myofascial syndromes and suggests that the most

important cause is sudden acute trauma to myofascial structures. Travell et al (1952) demonstrated that these trigger points are capable of producing specific patterns of referred pain described as myotomal, and that treatment of the trigger point abolishes this pain.

Simons (1975, 1976) has written a very comprehensive and important historical review of these muscle pain syndromes. He points out that the first significant contributions came from German medical literature at the turn of this century. Quoting from Simons' papers, a Dr. Muller (1910) stated that muscles afflicted with this muscular rheumatism are oversensitive to stretch and tension which produces pain, to cold which can trigger an acute attack, to momentary or sustained overexertion, to psychic stress, to poisons such as alcohol, and to prolonged inactivity, such as sleep. How little this picture has changed to this day.

He also quotes research by a Dr. Schmidt (1914) who suggested that these muscle hardenings were due to a neuralgia of the sensory muscle nerves. This was based on his observation of the pain sensitivity of the vertebral structures to pressure and on the favourable response to epidural injections of local anaesthetics in many patients with lumbago. He also implicated the muscle spindles as being responsible for the painfulness of the hardenings. A Dr. Shade (1919) biopsied rheumatic muscular hardenings detected before death in post mortem

muscles of war casualties. This hardening was noted to remain unchanged during anaesthesia. Histological examination showed only normal muscle.

He also refers to a paper by Port (1920) who stated that chronic (nodule) rheumatism can be recognised by the fact that it is caused by chilling, that its symptoms should wax and wane with the barometer, that one should be able to palpate within the muscle a local infiltration of unexplained pathology and that the pain and palpable findings should resolve with massage in all but the most long standing cases. A Dr. F. Lange (1925) recommended as treatment the use of a blunt instrument for brutal massage (Gelotripsie). A Dr. M. Lange (1931) distinguished muscle hardenings from callus by the tendencies of hardenings to be painful to digital pressure and responsive to massage. He also noted the subconscious attempt of the patient to minimise the pain by contracting the muscle being examined (i.e. the "jump sign"). Furthermore, he was the first to locate precisely and map out the commonly found muscle hardenings and related them to various painful symptom complexes, such as certain types of headache. He recommended traumatic massage, sometimes with follow-up self massage, to prevent recurrences.

Simons also refers to work by Ruhmann (1932) who reproduced painful muscle hardenings by injecting various substances into muscles. He concluded that these substances initiated sustained muscular contractions with

sufficient reflex excitation to obstruct outflow of waste products so that a vicious cycle was established. He thought that massage worked by improving the circulation.

Simons also mentions that Dr. H. Kraus (1937) was the first to employ ethyl chloride spray in the treatment of painful muscle hardenings, and Reichart (1938), who was the first to point out the myotomal referred pain patterns on stimulation of muscle hardenings. This preceded the American 'trigger point' school.

Regarding biopsy studies Simons quotes work by Dr. M. Lange (1931). Biopsies were taken from chronic muscle hardenings. These showed chronic degenerative changes within the hardenings. He also quotes work by Elliott (1944) who presented evidence for a neurogenic mechanism involved in some cases. Two patients with tender painful nodules were treated successfully with injections of procaine. Several months later they recurred, this time with evidence of nerve root pressure, proved at operation. Elliott did electromyographic studies of tender spots in buttock and calf muscles, using needle electrodes, in eight cases of sciatica which he attributed to irritation of nerve roots due to a prolapsed disc found at operation. In almost every case of deep tenderness, deep palpation of muscle close to the needle elicited bursts of motor unit activity in only that part of the muscle. He identified a localised hyper-irritability of the muscle in response to mechanical stimulus.

Simons also points out that Steindler and Luck (1938) first introduced the concept of referred pain from a distant pressure painful locus in ligament or muscle. These were, or course, first called trigger points by Travell and co-workers (1942), who also described the method of injecting the trigger points with procaine. They also noted the efficiency of sustained local pressure which was now called the Libman manoeuvre. They considered sustained spasm of the muscle itself a more likely cause than sympathetically mediated sustained spasm of the vessels supplying the muscle. Travell also described her own variation of the use of the ethyl chloride spray, combined with gentle passive stretch of the involved muscle. Travell also arranged several muscle biopsies of her trigger points. No pathological changes were detected. She also noted that the referred pain patterns from these trigger points were remarkable for the consistency of the location of the site of greatest intensity. She also noted that injections of normal saline into the trigger points were only slightly less effective than procaine, and that peppering the area with a dry needle was only slightly less effective than normal saline.

Simons points out that Travell theorised that protective splinting that keeps the initially injured muscle in the shortened position facilitates the extension of the disease process through a spasm-pain-spasm cycle. This painful shortening of skeletal muscle can remain a pathophysiological process for many years evidenced by

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Footnote: The papers referred to by Simons have not been personally examined.

the rapid return to normal function with treatment, in some long-standing cases.

The recommended treatment for these trigger points is varied. Physiotherapists have employed heat and deep friction to tender focal "Fibrositis" for many years. Travell et al (1952) recommend the use of coolant sprays, originally ethyl chloride, spraying the muscle from origin to insertion and combining this with passive stretch of the muscle. Travell makes the important observation that this technique does not work if the skin is cooled too much. Berges (1973) and others recommend injecting the trigger point with local anaesthetics. It is relevant that Judovich et al (1948) emphasised that treatment of what they called segmental neuralgia with injections of local anaesthetics should also include local injections to the segmentally related tender para-vertebral region. Some osteopaths and physicians (Gaymans 1973) employ "hold - relax" techniques. The muscle containing the trigger point is positioned just short of its full passive stretch and then made to isometrically contract for approximately seven seconds. On relaxing it is found that there is an increase in the range of passive stretch. This extra slack is then taken up and the isometric contraction repeated. It does not work if the contraction causes marked discomfort.

Dry needling (acupuncture) of these trigger

points is said to be effective. Electrical stimulation of the needles is thought to be even more effective. A recent study (Boas et al 1976) suggests that electrical stimulation of the skin over these trigger points, using what are called pain block machines (transcutaneous nerve stimulation) may be as effective as acupuncture. It is relevant that Melzack et al (1977) found a close correlation between the common sites of trigger points and the traditional acupuncture sites.

This poses the question: have all the different approaches to these painful syndromes something in common?

Earlier studies on electrical activity in the loci of muscle 'spasm' failed to demonstrate increased electro-myographic activity (Kraft et al 1968). A more recent study (Cobb et al 1975) seemed to show increased electrical activity in artificially induced muscle pain. These results are questionable because of the very high sensitivity of the recording equipment. There is no certain proof that the recordings did not come from elsewhere. Numerous authors have remarked on the difficulty and necessity of precisely locating the trigger point for successful procaine injection. This suggests that the trigger point itself may be a well innervated structure that occupies a small volume of muscle. Unless the electromyographer had accurately located the trigger point any conclusion he may come to would therefore be invalid.

Muscle biopsy is notoriously unreliable, but according to Berges (1973) sampling from these 'fibrositic' areas show variations, from normal muscle to histological changes denoting muscle degeneration and fibrosis.

It is likely that these varying reports are related to the length of time that the trigger point has been present. Clinical experience leaves no doubt about their existence. The advocates of different treatments all note that those of recent onset may disappear after only one treatment whereas those that have been present for some time may need several treatments. They do not cause continuous pain. According to Berges and others, they may be initiated by chilling, muscle fatigue, arthritis, or other musculoskeletal disorders, and rarely, by visceral disease. They feel like knots of muscle spasm that are extremely tender. When treated by direct firm pressuring they are very painful for about two to three minutes. The pain then goes and the knot of muscle melts away. Likewise they disappear after injections of local anaesthetics.

It is a relevant observation that these trigger points always tend to recur at the same place in each individual, and even when not giving rise to any symptoms they can still be palpated.

The absence of evidence of electrical activity

in these trigger points could be explained by the obvious fact that there is no extra activity in the resting state. It is possible that the act of palpation provokes the spasm. Caccia et al (1973) have shown that certain skin mechanoreceptors when stimulated have an excitory effect on the motor neurones. They also note a late inhibitory response which they think is due to stimulation of the larger group $\overline{11}$ mechanoreceptor fibres, and that this late response may operate through inhibition of the fusimotor system.

The fact that pressuring can disperse these trigger points is paradoxical. It is a painful procedure and stimulation of the nociceptor nerves leads to reflex excitation of the alpha motor neurones via a polysynaptic pathway (Wyke 1976). There is also an increase in gamma discharge on nociceptor stimulation. Granit (1975) called this a nociceptor reflex partnership. The nociceptors are also non adaptive, thus one would expect that pressuring would aggravate rather than relieve the pain. It is a common experience that two to three minutes of constant firm pressure is needed before the pain goes and the knot of 'muscle spasm' melts away. As the patient improves it takes less time and is less painful. Also, it is a relevant observation that when there is a disc prolapse that has led to an inflammatory response pressuring of associated trigger points in the gluteal muscles aggravates rather than relieves the pain. The pressuring may

excite powerful inhibitors of nociceptor input. This is unlikely as receptors such as the paccinian corpuscles are rapidly adapting (Mountcastle 1974). The pressuring may eventually have some inhibitory effect on the inflow from the nociceptors by interfering with the blood flow to the area. This would reflexly reduce the excitability of the motor neurones.

The perception of pain is not the inevitable result of stimulation of nociceptor nerve endings. There is evidence to suggest that accurate localisation of pain depends on inflow from other mechanoreceptors (Wyke 1976). The so-called 'slow pain' fibres, thought to be the smaller slower conducting nociceptors, and which make up about 70% of the total nociceptor input, have multiple connections with interneurones in their passage to the higher centres. Their input can thus be influenced at several levels.

It is well known that at the spinal level stimulation of the larger high threshold mechano-receptors has an inhibitory effect on nociceptor input. This knowledge has been utilised in the management of some painful syndromes. To explain this phenomena Melzack and Wall (1965) proposed a 'gating' mechanism whereby input from large mechanoreceptors stimulated cells in the substantia gelatinosa which effectively block the transmission of impulses from the nociceptors to the

transmission cells. This theory is no longer acceptable to neurophysiologists. It is thought that collaterals from the larger cells have an inhibitory effect at some stage in the transmission of small fibre input.

The possible role of disordered muscle activity in the perpetuation of spinal pain has received scant attention in the medical literature. Schlesinger et al (1951) demonstrated that in some cases of acute low back pain with limitation of the straight leg raising test, an intravenous injection of myenesin (a muscle relaxant acting mainly on the higher polysynaptic reflex arcs) caused an immediate increase in the straight leg raise, which persisted, and, when followed by conservative treatment, led to long term cure. This implies that in these cases the main cause of the symptoms was muscle spasm. There is current enthusiasm for the surgical procedure devised by Skyrm Rees (1971) which he calls 'multiple bilateral subcutaneous rhizolysis of segmental nerves.' This was claimed to de-ennervate the facet joint capsule but recent studies show this to be untrue (Bogduk et al 1977. King et al 1976). What is obvious from these studies is that the pain relief is dependent upon the de-ennervation of muscle which contains a trigger point.

King et al (1976) used a radio frequency current which destroys tissue in an area of approximately 7 x 11mms.

This current is passed through a needle that is inserted into the area of maximal tenderness in the paravertebral muscles (trigger point). They made the important observation that in one particular case there was an immediate relief of pain in the back and leg and loss of painful limitation of straight leg raising. The symptoms returned at a later date and at operation a large disc protrusion was found. A similar observation has been made on a patient after manipulation of the lumbar spine (Fisk 1975).

That disordered muscle activity could play an important role in the perpetuation of pain in the back seems theoretically possible when the characteristics peculiar to the spine are considered:

In the lumbar and cervical spine the segmental nerve supply overlap at least three segments and also cross the mid line. The main muscles overlap several segments and receive their nerve supply from several segments. There is the close proximity of each spinal joint. Thus, if one joint was strained the overlying muscles could be receiving conflicting information from several normal and one abnormal joint.

Denslow et al (1947) inserted recording electrodes into the paravertebral muscles at varying levels of the dorsal spine in normal resting subjects and recorded the amount of pressure (using a specially designed pressure meter) needed to produce muscle activity. The pressure

was imposed on the spinous processes. The results showed that the required pressure varied considerably not only at different levels but also from side to side. They argued that pools of motor neurones, even in close proximity, showed varying degrees of closeness to threshold activity in the resting state, and that their pressure meter gave a measure of this reflex excitability. They also argued that it was likely that the facilitating impulses arose from segmentally related structures.

The pressure could be stimulating mechanoreceptors in the skin which have reflex connections with the motor neurones, but why the different response recorded on the two sides? The pressure could also be affecting the mechanoreceptors in the spinal joint complex which also have a reflex connection with the motor neurones.

Unlike the lumbar and cervical spine the nerve supply to the dorsal spinal joint complex is strictly segmental (Wyke 1970). The mechanoreceptors within the capsule of the apophyseal joints have been studied in detail by Wyke (1972). He points out the profound effect of stimulation of these receptors on the overlying muscles. The Type 1 receptors are of particular importance. They are small myelinated (6 - 9 μ) and are classified as static and dynamic mechanoreceptors. They have a low threshold and are slowly adapting. Like the gamma motor

nerves they show continuous activity, and exercise a continuous (tonic) influence over the reflex regulation of the tone of the muscles operating over the joint (Wyke 1967). The precise nature of this reflex pathway is not fully understood but Wyke suggests that it is more likely to be a polysynaptic projection to the gamma (fusimotor) rather than the alpha motor neurones.

It is also of relevance to mention the Type $\overline{111}$ receptors. They are the articular homologue of the Golgi tendon organ and are structurally identical (Wyke 1967). They are high threshold slowly adapting end organs and only come into play at the extremes of joint movement. They are stimulated by joint distraction (manipulation) and strong traction. They also have a profound influence on overlying muscle activity, which is mainly inhibitory.

It would be an impossible task to survey the literature on the alpha and gamma motor activating system, the spinal and supraspinal control and co-ordination of movement and the functions of the muscle spindles. Most of the neurophysiological research has concentrated on the spinal reflex level in lower order animals. Little is known about supraspinal control and monitoring of movement in man. The following research is relevant to this thesis:

Hunt (1951) showed that in the spinal cat there

is a background gamma discharge to 'resting' muscle. He thought that in normal function a combined afferent discharge from skin, muscles and probably other deep structures will regulate the small nerve activity to muscle. He thought that the pattern of skin stimulation may be of particular importance.

Hunt et al (1958) again pointed out the marked influence of cutaneous stimulation on gamma motor nerve excitability and that there was no direct reflex connection between the spindle afferents and the gamma motor nerves. They thought that the gamma nerves were innervated by polysynaptic pathways within the spinal cord.

Granit (1964) pointed out that increased gamma activity drives the alpha motor neurones, and that intrafusal muscles can control the spindle length, therefore it can be given a length setting or gamma bias, which is one of the biological mechanisms that may go wrong. He also made the point that the act of discharging is delicately poised in the alpha motor neurones, and when the interneurones are damped down by pentobarbital sodium the motor neurones fail to discharge tonically, yet they can be heavily bombarded by impulses from stretch receptors. No motor neurone is likely to go off from the muscular end alone unless the input is synchronised as in tendon and H reflexes, which are relatively primitive functions. Also, both spindle secondaries and golgi

tendon organs go to interneurones whose state of excitability will determine whether or not they will exercise their effect on the motor neurones.

Buchwald (1967) showed that stimulation of the deep pressure receptors (Group 2), such as the secondary muscle spindle afferents, inhibit extensors and facilitate flexors, via polysynaptic connections (i.e. the withdrawal or flexor reflex).

Eldred (1967) thought that co-ordination of alpha motor and fusimotor outflow probably took place in the cerebellum.

Szumski (1967) stated that the gamma system was ideally situated to function as a "motor activating or alerting system." It is a low threshold motor system extensively influenced by supraspinal areas which function to modulate afferent muscle spindle activity.

Taylor (1970) thought it possible that the strength of fusimotor drive could be modified during the learning of a movement, so that the distribution between the two routes may be optimised at the point where no change in the spindle output occurs if the movement is correctly executed.

Vallbo (1970) demonstrated a central co-activation of fusimotor and alpha motor fibres during a

willed voluntary contraction in humans. Matthews (1971) stated that the understanding of the muscle spindle is likely to be an essential prelude both for understanding the normal control of movement and for understanding the derangements of function underlying spastic and hypertonic states. He also pointed out that there are two to three times as many static gamma fibres than dynamic gamma fibres, the former going predominantly to the nuclear chain fibres and the latter to the nuclear bag fibres. There is also adequate evidence that the central nervous system can exert independant control over the static and dynamic fusimotor fibres. He also pointed out that the independence of the fusimotor pathway from the ordinary motor pathway provides the essential degree of freedom which would allow the fusimotor discharge to be appropriate primarily to the trajectory of the planned movement, while the direct alpha discharge in addition has to be appropriate for the external load. This is a "servo-assistance" of movement rather than a "follow-up length servo" system.

Vallbo (1974) studied muscle spindle discharge during voluntary isometric contractions and noted the close co-ordination between spindle frequency and torque. This suggests a parallel modulation of the alpha and gamma outputs during voluntary contractions. The dominant type of gamma outflow was of the static variety. He pointed out that the muscle spindles and their central

connections do not constitute a very powerful mechanism for holding the muscle at a constant length when the load varies.

Inbar (1975) pointed out that the gamma system is an efficient agent for carrying out modifications in the dynamics of the muscle reflex system. He called this an optimal adaptive system concept. He also stated that volitional muscular control is executed principally through the alpha system.

Granit (1975) states that the gamma motor neurones maintain the spindles in a state of responsiveness to length and to rate of change of length of muscle. Centrifugal control of muscle spindles takes two forms; first, a steady and slowly fluctuating tonic firing of fusimotor neurones, as a part of a general state of arousal or readiness to move, independent of the firing of alpha motor neurones and not related in time to specific movements; secondly, a precise co-activation of alpha and gamma motor neurones which is related to the time-course of specific movements. Both types are likely to be important in man. Hagbarth et al (1975) postulate that fast movements are initiated directly through alpha motor neurones and that gamma loop influence during alternating movements helps to keep flexor and extensor muscles working in a regular reciprocal fashion, with contractions adjusted in strength to the external loads.

Nelson (1976) showed that fusimotor blockade led to overestimation of force requirements and increased error in production of predetermined pattern of motor control.

Fromm et al (1976) showed that primary, and probably secondary, afferents of the pretibial flexors inhibit the extensor gamma efferents. They thought that the probable pathway was via an IA inhibitory interneurone. They note that the only difference between the alpha and gamma efferents is the monosynaptic reflex, but that a polysynaptic connection between the spindle afferents and the gamma efferents has been detected.

In summary, the gamma system is a constantly active monitoring and activating system that may be influenced at various levels of the neuraxis. In back problems one of the key levels could be the tie-up with the constantly active Group I joint mechanoreceptors. It is of interest that Korr (1974) suggested that local increase in gamma activity could be responsible for the so-called osteopathic lesion.

REFERENCES

Berges, P. U. (1973) Myofascial pain syndromes. Postgrad. Med. 53 : 161 - 168

Boas, R. A., and Hatangdi, V. S. (1976). Electrical stimulation in the relief of pain : A pilot study. N.Z. Med. J. 84 : 230 - 233.

Bogduk, N., Colman, R.R.S., Winer, C. E. R. (1977). An anatomical assessment of the "Percutaneous Rhizolysis" procedure. Med. J. Aust. I : 397 - 399

Bourdillon, J. F. (1973). Spinal Manipulation. 2nd Ed. London : Heinemann. p. 112 - 116

Buchwald, J. S. (1967). Exteroceptive reflexes and movement. Am. J. Phys. Med. 46 : 121 - 128

Caccia, M. R., McComas, A. J., Upton, A. R. M., Blogg, T. (1973) Cutaneous reflexes in small muscles of the hand. J. Neurol. Neurosurg. & Psych. 36 : 960 - 977

Cailliet, R. (1968) Low Back Pain Syndrome. 2nd Ed. p. 42 - 44. Philadelphia : Davis.

Cobb, C. R., deVries, H. A., Urban, R. T., Luekens, C. A., Bagg, R. J. (1975) Electrical activity in muscle pain. Am. J. Phys. Med. 54 : 80 - 87

Crisp, E. J. (1960) Disc Lesions and other Intervertebral Derangements. p. 43 - 44 Edinburgh : Livingstone.

Cyriax, J. (1962) Text-book of Orthopaedic Medicine. Vol. 1. 4th Ed. p. 56 - 57. London : Cassell.

Denslow, J. S., Korr, I. M., Krems, A. D. (1947) Quantitative studies of chronic facilitation in human motoneuron pools. Am. J. Physiol. 150 : 229 - 238.

Dillane, J. B., Fry, J., Kalton, G. (1966) Acute back syndrome - a study from general practice. Brit. Med. J. 2 : 82 - 84.

Edgar, M. A., Nundy, S. (1966) Innervation of the spinal dura mater. J. Neurol. Neurosurg. & Psych. 29 : 530 - 534.

Eldred, E. (1967). Functional implications of dynamic and static components of the spindle response to stretch. Am. J. Phys. Med. 46 : 129 - 140.

Elliot, F. A. (1944). Tender muscles in sciatica : electromyographic studies. Lancet. I : 47 - 49.

Farfan, H. F. (1973) Mechanical Disorders of the Low Back Philadelphia : Lea & Febiger. p. 145.

Feinstein, B., Langton, J., Jameson, R., Schiller, F. (1954). Experiments on pain referred from deep somatic tissues. J. Bone & Joint Surg. 36A : 981 - 997

Fisk, J. W. (1975). The straight leg raising test - its relevance to possible disc pathology. N.Z.M.J. 81 : 557 - 560

Fisk, J. W. (1977). Approaches to the Validation of Manipulation Therapy.
Ed. Buerger, A. A. & Tobis, J. S.
Springfield, Illinois : Thomas.
p. 236 - 270.

Fisk, J. W. (1977A) A Practical Guide to Management of the Painful Neck and Back. p. 94 - 102
Springfield, Illinois : Thomas.

Fromm, C., Noth, J., Thilmann, A. (1976).
Inhibition of extensor gamma motorneurones by antagonistic primary and secondary spindle afferents.
Pflugers Archiv. 363 : 81 - 86.

Gaymans, F. (1973) Neue Mobilisations. Prinzipien und technikan an der. WS.
Manuelle Medizin. 2 : 6 - 11.

Ghormley, R. K. (1951). An aetiological study of backache and sciatic pain.
Proc. Staff Meet. Mayo Clin.
26 : 457 - 463.

Granit, R. (1964). The gamma loop in the mediation of muscle tone.
Clin. Pharm. & Therapeut.
5 : 837 - 847.

Granit, R. (1975) The functional role of the muscle spindles : facts and hypotheses.
Brain. 98 : 531 - 556.

Hagbarth, K. E., Wallin, G., Lofstedt, L. (1975).
Muscle spindle activity in man during voluntary fast alternating movements.
J. Neurol. Neurosurg. & Psych.
38 : 625 - 635.

Hirsch, C. (1956) The mechanical response in normal and degenerated lumbar disc.
J. Bone & Jt. Surg. 38A : 242 - 243

Hockaday, J. M., Whitty, C. W. M. (1967).
Patterns of referred pain in the normal subject.
Brain. 90 : 481 - 496.

Hunt, C. C. (1951)
The reflex activity of mammalian small-nerve fibres.
J. Physiol. 115 : 456 - 469.

Hunt, C. C., Paintal, A. S. (1958).
Spinal reflex regulation of fusimotor neurones.
J. Physiol. 143 : 195 - 212.

Inbar, G. F. (1975).
Modulation of dynamic parameters of muscle reflex by selective activation of the gamma system.
Biol. Cybernetics. 19 : 169 - 180.

Judovich, B., Bates, W. (1949).
Pain Syndromes, 3rd Ed. p. 52 - 62
Philadelphia : Davis.

Kellgren, J. H. (1939).
On the distribution of pain arising from deep somatic structures with charts of segmental pain areas.
Clin. Sci. 4 : 35 - 46.

King, J. S., Lagger, R. (1976)
Sciatica viewed as a referred pain syndrome.
Surg. Neurol. 5 (1) : 46 - 50.

Korr, I. M. (1974).
Proprioceptors and the behaviour of lesioned segments.
Osteopathic Annals. August 12 - 33

Kos, J.,Wolf, J. (1971)
Intervertebral menisci and their possible role in vertebral blockages.
From the Report of the 3rd International Congress of Manual Medicine.
Monaco. 14 - 17 Oct.

Kraft, G. H., Johnson, E. W., La Ban, M. M. (1968).
The fibrositis syndrome.
Arch. Phys. Med. & Rehab.
49 : 155 - 162.

Kraus, H. (1967) Prevention of low back pain.
J. Occup. Med. 9 : 555 - 559.

Lewin, T. (1965). Osteoarthritis in lumbar synovial joints.
Acta Orthop. Scand. Supp. 73.

Matthews, P. B. C. (1971).
Recent advances in the understanding of the muscle spindle.
Scientific Basis of Med. 99 - 128.

Melzack, R., Stillwell, D. M., Cox, E. J. (1977).
Trigger points and acupuncture points for pain : correlations and implications.
Pain. 3 : 3 - 23

Mountcastle, V. B. (1974).
Medical Physiology. 13th Ed.
Vol. I.
Ed. by Mountcastle, V. B. p. 288.
Saint Louis : Mosby.

Nachemson, A. (1976). The Lumbar Spine and Back Pain.
Ed. Jayson, M. p. 355 - 366.
New York : Grune & Stratton.

Nelson, A. J. (1976). Fusimotor influence on performance of ankle dorsiflexors in young adults.
Physiotherapy. 62 : 117 - 122.

Pedersen, H. E., Blunck, C. F. J., Gardner, E. (1956).
The anatomy of lumbosacral posterior rami and meningeal branches of spinal nerves (sinu vertebral nerves).
J. Bone & Jt. Surg. 38A : 377 - 391.

Schlesinger, E. B., Stinchfield, F. E. (1951).
The use of muscle relaxants as an aid in the diagnosis and therapy of acute low back disorders.
J. Bone & Jt. Surg. 33A : 480 - 484

Simons, D. G. (1975) Muscle pain syndromes. Part I.
Am. J. Phys. Med. 54 : 289 - 311

Simons, D. G. (1976) Muscle pain syndromes. Part 2.
Am J. Phys. Med. 55 : 15 - 42.

Skyrme Rees, W. E. (1971).
Multiple bilateral subcutaneous rhizolysis of segmental nerves in the treatment of the intervertebral disc syndrome.
Ann. Gen. Pract. 16 : 126 - 127.

Stoddard, A. (1969). Manual of Osteopathic Technique.
p. 27.
London : Hutchinson.

Szumski, A. J. (1967) Mechanisms underlying normal motor behaviour.
Am. J. Phys. Med. 46 : 52 - 68.

Taylor, A. (1970). Muscle receptors in the control of voluntary movement.
Proc. of the 2nd Sci. Meet. of the International Med. Soc. of Paraplegia. Edinburgh 27 - 28th July. 167 - 172.

Travell, J., Rinzler, S. H. (1952).
Scientific exhibit : the myofascial genesis of pain.
Postgrad. Med. II : 425 - 434.

Unsworth, A., Dowson, D., Wright, V. (1971).
Cracking joints. A bioengineering study of cavitation in the metacarpophalangeal joint.
Ann. Rheum. Dis. 30 : 348 - 358.

Vallbo, A. B. (1970). Central co-activation of fusimotor and ordinary motor fibres during a willed voluntary contraction. Acta Physiol. Scand. 78 : 315 - 333

Vallbo, A. B. (1974). Human muscle spindle discharge during isometric voluntary contractions. Amplitude relations between spindle frequency and torque. Acta Physiol. Scand. 90 : 319 - 336.

Wyke, B. (1967) The neurology of joints. Anns. Roy. Coll. Surg. Eng. 41 : 25 - 50.

Wyke, B. (1970). The neurological basis of thoracic spinal pain. Rheum. & Phys. Med. 10 : 356 - 366.

Wyke, B. (1972). Articular neurology - a review. Physiotherapy. 58 : 94 - 99.

Wyke, B. (1976) The Lumbar Spine and Back Pain. Ed. Jayson, M. 189 - 256. New York : Grune & Stratton.

1. THE PASSIVE HAMSTRING STRETCH TEST: CLINICAL EVALUATION

INTRODUCTION

Attempts to assess the effectiveness of any treatment for low back pain problems have been handicapped by the lack of objective measurable clinical signs. Stimulation of the pain receptors in the deeper structures of the back leads to diffuse tenderness, restricted movement, focal muscle spasm, often at a distance from the site of stimulation, and sometimes areas of surface hyperalgesia. These are all difficult to assess objectively.

One measurable clinical sign that has been used since it was first described by Forst, in 1881 (Cyriax, 1969), is painful restriction of straight leg raising. A modification of this test which estimates the amount of passive stretch available in the hamstring muscles is well known to Osteopaths but little known to Allopathic physicians. To assess this degree of passive stretch the palm of one hand is placed over the contra-lateral anterior superior iliac spine (the centre of the palm being that which is most sensitive to a change of movement), and the leg is raised with the other hand until the pelvis is felt to rotate, as described by Mitchell (1973).

In a proportion of patients suffering from low back pain it has been observed that there is a significant difference in the available passive hamstring stretch on the two sides, and that manipulation of the lumbo-sacral region of the spine eliminates this difference (Fisk 1977A). A recent pilot study in the format of a single blind controlled trial (Tobis et al, 1977) suggested that rotational manipulation of the lumbo-sacral region of the spine was superior to placebo treatment at a statistically significant level, using alteration in the passive hamstring stretch test as the criterion of improvement. This trial is of considerable importance because it is the first to point to the superiority of manipulation over placebo treatment, and could point to avenues of research into the mechanisms of back pain and how it may be relieved.

In this trial the measurements of the passive hamstring stretch were taken by the same observer. Before further research takes place it is important to demonstrate that different observers can make the same independent measurements in the same patient.

METHOD

The technique for testing the degree of passive hamstring stretch was demonstrated to three physiotherapists,

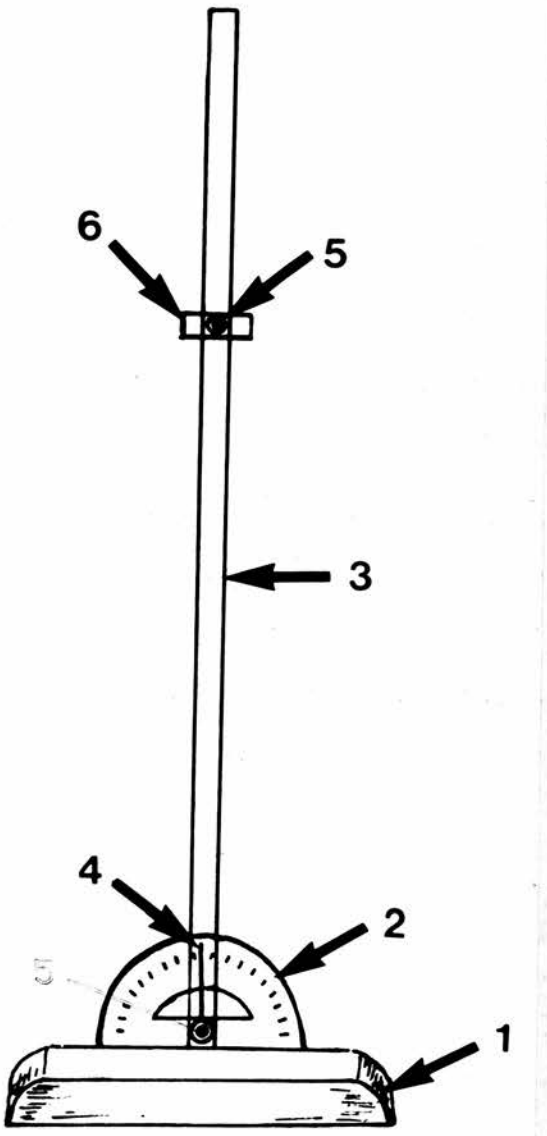
working in the Department of Physical Medicine and Rehabilitation, Waikato Hospital. They were asked to practice the test as often as possible, until they felt confident in their ability to sense the beginning of rotation of the pelvis.

A modified goniometer was constructed, as shown in the diagram (see Fig. 1). The locking screw was positioned so that it was level with the patient's greater trochanter. The adjustable perspex block was positioned so that its proximal edge was in contact with the undersurface of the lateral malleolus. The distal part of the arm of the goniometer was marked off in centimeters. It was thus possible to record the position of the adjustable block for each patient, making a note of the position on the records.

The examiner raised the leg slowly and smoothly until rotation of the pelvis was sensed. The leg was held in this position whilst an assistant elevated the goniometer arm until the upper surface of the adjustable block was resting on the undersurface of the patient's lateral malleolus. The angle made by the arm of the goniometer with the horizontal was then recorded.

Three patients known to have relatively tight hamstrings were then examined consecutively and independently by the three physiotherapists. An assistant

FIG. 1



MODIFIED GONIOMETER

1. Wooden Base 38 cm long
   5 cm high
   10 cm thick
2. Plastic Transparent Protractor
   20 cm diameter
3. Aluminium Arm 115 cm long
   $2\frac{1}{2}$ cm wide
   - Graded in cm at the distal end
4. Red Line Painted Down the Centre of the Arm
5. Locking Screws
6. Plastic Sliding Block 7 cm high
   2 cm wide
   3 cm thick

FIG 2

EXAMINERS

		Mrs. C.B		Mrs. H.A.		Mr. P.J.	
		L	R	L	R	L	R
Patient 1	1st reading	58°	62°	56°	62°	54°	62°
	2nd reading	59°	64°	57°	62°	53°	61°
	3rd reading	58°	63°	56°	62°	54°	62°
	Average	58°	63°	56°	62°	54°	62°
Patient 2	1st reading	55°	56°	58°	60°	55°	52°
	2nd reading	55°	56°	60°	58°	55°	55°
	3rd reading	55°	57°	55°	56°	57°	57°
	Average	55°	56°	57°	58°	55°	55°
Patient 3	1st reading	45°	50°	44°	49°	45°	50°
	2nd reading	45°	51°	47°	46°	46°	51°
	3rd reading	47°	51°	44°	49°	43°	51°
	Average	45°	50°	45°	48°	45°	51°

measured the angles and recorded three consecutive measurements for each leg, without informing the examiner of the results. Each examiner was unaware of the measurements of the other two, and was not in the room at the time they were being taken.

RESULTS

See Fig. 2.

DISCUSSION

These results demonstrate the objective value of the passive hamstring stretch test as a potential research tool, the maximum difference in the averages of the three independent examiners being only three degrees.

During trials of this test certain difficulties have become apparent. Every so often the readings of one of the independent examiners has been up to 7° different to the other two. It is thought that the rate at which the leg is raised plays a part in these occasional differences. It has also been found that a too rapid repetition of the test leads to an alteration in the available passive stretch in the hamstrings. One minute intervals between tests seems to eliminate this possibility. Some patients are quite incapable of consciously relaxing enough to make the test possible. It is, of course,

imperative for the patient to keep the knee straight. It is important to keep the rate of raise fairly constant. If the examiner has doubt about the point when the pelvis is beginning to rotate the test should be started again. If at any point in the arc the movement is stopped and the leg is then waggled up and down reflex activity in the hip extensors is provoked (probably a monosynaptic stretch response) and movement will be sensed by the examining hand.

With the patient lying supine, passive elevation of one leg to the point where the pelvis begins to rotate on the opposite side must be a measure of the available stretch in the hip extensors. These are mainly the gluteus maximus, the hamstrings, and to some extent the adductor magnus. The main body of the hamstrings crosses both the hip and knee joints. Flexion of the knee relaxes the hamstrings. If the hip is flexed with the knee also flexed rotation of the pelvis does not take place until the thigh is at least at a right angle to the trunk. Thus, the detection of the angle where pelvic rotation begins to take place whilst passively raising the leg with the knee kept straight must be a measure of the available passive stretch within the hamstring muscle group.

In the candidate's experience, a difference of approximately 10^{o} between the two sides may be of significance, in patients with low back problems. A difference

of this magnitude is found in approximately 10% of the candidate's patients who suffer from low back problems. It is impossible to stipulate a detailed clinical picture characteristic of such patients, but they usually have some or all of the following symptoms and signs in common:

The low back ache tends to favour one side. This deep ill-defined ache usually spreads into one buttock. This can spread into the back of the thigh, and there may be a patch of discomfort in the back of the calf. The patient usually wakes up stiff and sore. This morning soreness quickly passes off with exercise. Bending, of course, aggravates the ache. Standing and lying relieves the discomfort. So does sitting, but the ache returns after sitting for any length of time. Sitting for any length of time in a car is particularly uncomfortable. Walking may aggravate the discomfort. This would be explained by the fact that the hip extensors are inactive whilst standing but both are active during different phases of the walking cycle (Basmajian, 1974).

Examination reveals characteristic clinical findings:

(a) Whilst standing, the patient is asked to bend forward as far as he or she can without undue discomfort, in an attempt to touch the toes with the knees straight. Palpation of the posterior superior iliac spines reveal that the spine on the affected side is held back.

If the patient is then asked to sit on a stool and attempt to bend forward and put his or her head between the knees, palpation of the posterior spines reveals that they now move synchronously.

(b) Palpation of the gluteals invariably reveals a trigger area (Travell & Rinzler, 1952) which exhibits the "jump sign" (firm pressure on the area makes the patient jump).

(c) There is usually focal tenderness over the supra-spinous ligament between the 5th lumbar spine and the sacrum, with associated focal paravertebral muscle spasm and tenderness which is usually more marked on the affected side.

(d) The passive hamstring stretch test reveals marked restriction on the affected side.

The possible significance of these findings has been discussed elsewhere (Fisk, 1977 B). When the patient consciously relaxes the leg and the hamstrings are then put on the stretch by passively flexing the thigh, with the knees kept straight, the amount of allowable stretch will be determined by the sensitivity of the stretch receptors within the muscle (i.e. the muscle spindles). These will be the only active stretch receptors in the absence of tension within the muscle. That it is possible to suddenly alter the range of this test by manipulation of the lower lumbar spine could, and probably does, imply that the sensitivity of the spindle has been altered in some way. The most likely explanation would be by a reflex alteration in the gamma inflow to the spindle. It has been suggested

as an alternative explanation that the manipulation frees up the sacro-iliac joint, thus increasing the range, but the initial examination of the patient shows that the sacro-iliac joint on the affected side is already showing more movement than its opposite number, and that they both move synchronously after manipulation. Furthermore, it has been found that treatment of the trigger area in gluteals, either by injections of local anaesthetic or by the old fashioned pressuring, will also effectively alter the degree of stretch on the affected side.

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Mrs. H. Allis. Senior Physiotherapist.
Mrs. C. Beresford. Senior Physiotherapist.
Mrs. N. Dawson. Physiotherapy Tutor.
Mr. P. Jarvie. Physiotherapist.

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REFERENCES

Basmajian, J. V. (1974) Muscles Alive 3rd ed. p. 253 - 255. Baltimore, Williams & Wilkins.

Cyriax, J. (1969) Textbook of Orthopaedic Medicine Vol. 1 5th ed. p. 451 - 457. London, Bailliere, Tindall & Cassell.

Fisk, J. W. (1977A) Approaches to the Validation of Manipulation Therapy Eds. Buerger & Tobis p. 236 - 270. Springfield, Illinois; Charles C. Thomas.

Fisk, J. W. (1977B) Practical Guide to Management of the Painful Neck and Back p. 65 - 67, 144. Springfield, Illinois; Charles C. Thomas.

Mitchell, F. L. (1973) An Evaluation & Treatment Manual of Osteopathic Manipulative Procedures p. 171 Institute for Continuing Education in Osteopathic Principles (ICEOP) Kansas City, Missouri

Tobis, J. S., & Buerger, A. A. (1976) Rotational Manipulation of the Lumbosacral Paravertebral area effect on straight leg raising tests. Arch. Phys. Med. Rehabilitation 57, 565.

Travell, J. & Rinzler, S. H. (1952). Scientific Exhibit: The Myofascial Genesis of Pain. Postgrad. Med. 11 p 425 - 434

2. THE PASSIVE HAMSTRING STRETCH TEST: A COMPARISON OF CLINICAL ESTIMATES WITH TENSION GAUGE MEASUREMENTS

INTRODUCTION

The possible value of the passive hamstring stretch test as a research tool in certain low back pain syndromes has been discussed. A chance observation by the candidate showed that the point at which movement was palpable in the contralateral anterior superior iliac spine coincided approximately with a marked increase in tension in the leg being raised. The tension was measured by incorporating a spring balance into a pulley system which was used to elevate the straight leg passively. If this can be confirmed human observer error would be eliminated from the passive hamstring stretch test. This would provide an objective research tool.

Ten normal subjects with measurably tight hamstrings were examined. The clinically determined end point of the passive hamstring stretch test was compared with measurements of the tension in the examined leg.

METHOD

A pair of modified boots were made (see Fig. 3). These allow the legs to be hooked up to a tension gauge

LEGEND

FIG 3

Modified boot:

a. Alloy backslab with plasterxeote covering.

b. c. Valcro fastening straps.

d. Adjustable arm with locking screw.

e. Linear tension gauge (Salter).

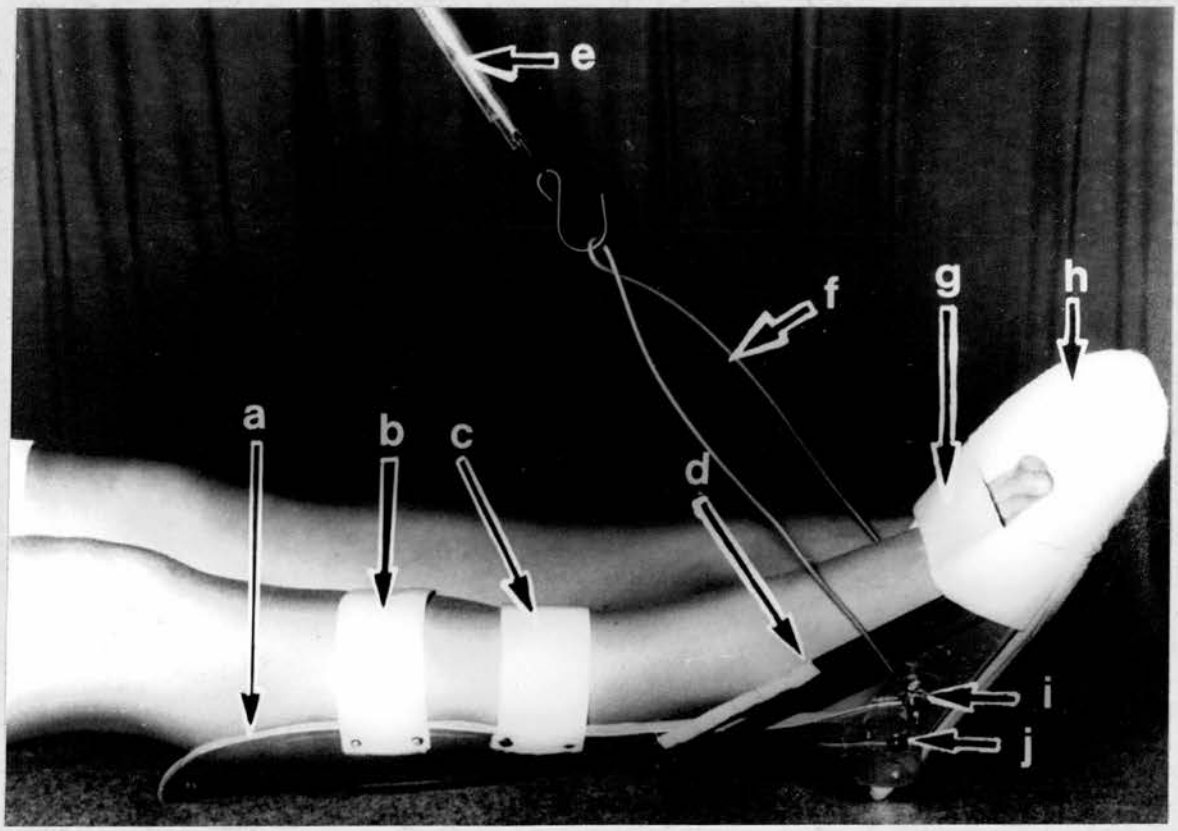
f. Freely pivoting loop of wire.

g. Valcro fastening straps for foot.

h. Footplate with crepe bandage covering.

i. Freely pivoting attachment of wire loop.

j. Pivot joint between backslab and footplate.

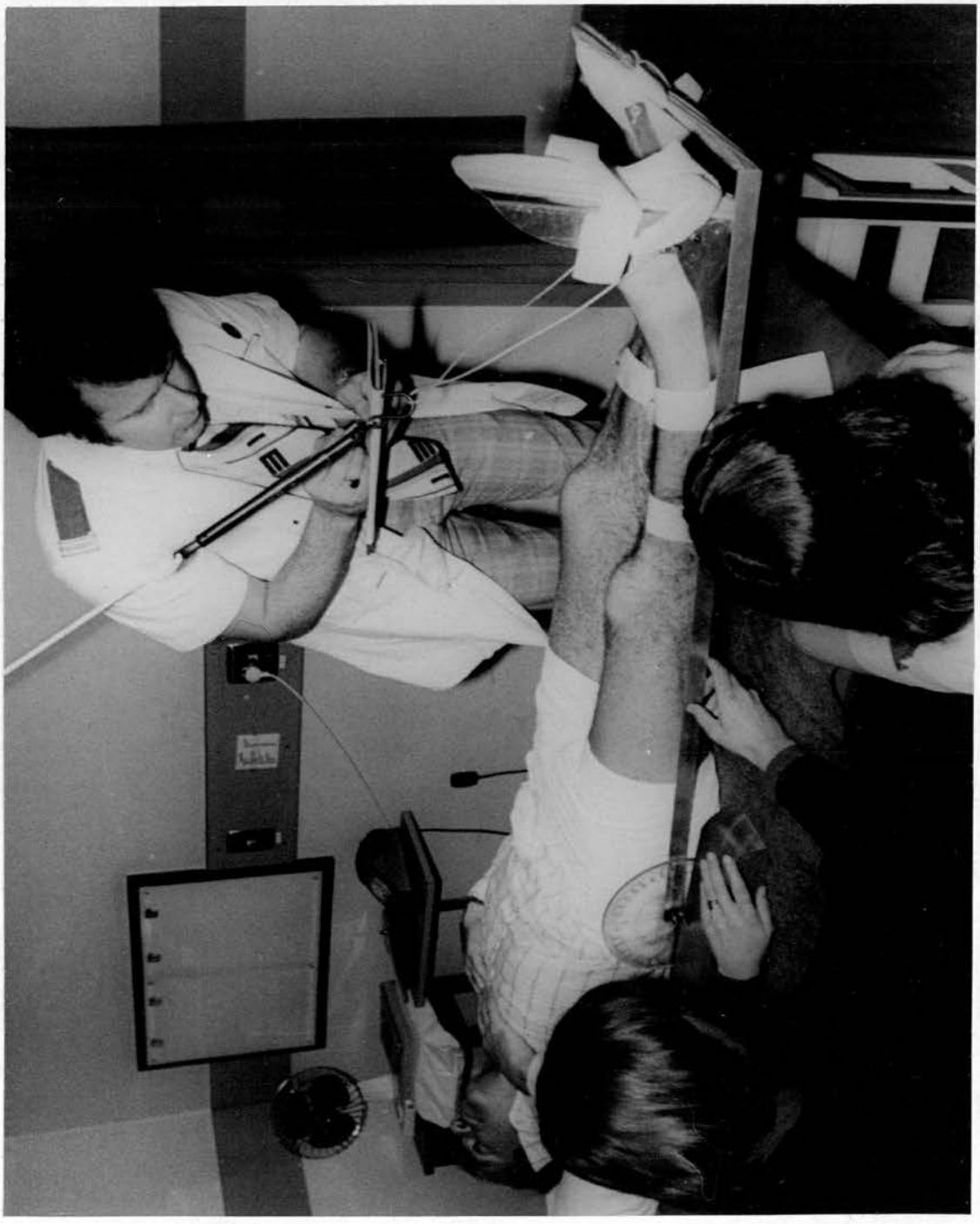


and pulley system, and for the ankle joints to be locked at any particular angle. These were fitted on the supine subject. The modified goniometer, minus the sliding perspex block, was placed so that the centre screw was positioned at the tip of the greater trochanter. The position of the calibrated long arm of the goniometer in relation to a fixed point on the heel plate of the boot was recorded. The goniometer could thus always be replaced in exactly the same position.

The subject was instructed to relax the leg and to keep the knee straight. The leg was raised smoothly by the candidate to the point where movement was sensed by the palpating hand on the contralateral anterior superior iliac spine. An assistant then raised the long arm of the goniometer to a predetermined point on the heel plate. Another assistant noted and recorded this angle between the long arm and the horizontal, reading from the protractor. Neither the subject nor the candidate could see the measurement that was recorded. Three consecutive measurements were made on each leg.

A Salter spring tension gauge was now hooked onto one of the boots (see Fig. 4). The rope attached to the gauge passed through a fixed pulley attached to the ceiling 185 cms. above the couch. The subject was positioned so that this pulley was over the sagittal plane. In most of the studies, depending on how tall the subject

FIG. 4



was, the distance from the vertical dropped from the pulley to the heel plate was 140 cms. This ensured that over the critical range of straight leg raising the angle of pull was close to a right angle to the leg.

The candidate took no further part in the experiment except to pull on the rope which elevated the leg. The goniometer was positioned as for the clinical estimate. One assistant raised the arm of the goniometer in five degree increments. The author pulled on the rope raising the leg till it was level with the goniometer arm. A second assistant checked that the level was correct, signalling the candidate when to stop. A third assistant, who was not present whilst the original clinical measurements were being taken, noted and wrote down the tension gauge measurements after each five degree raise.

The measurements were discontinued when the assistant had noted a consistent marked elevation of tension. This coincided with the subject experiencing increasing discomfort mainly behind the knee.

RESULTS

See Fig. 5.

The tension is measured in Kilograms.
In order to simplify the table the first six readings are missed out.

The results may be better illustrated in the form of a graph; see Fig. 6.

FIG 5

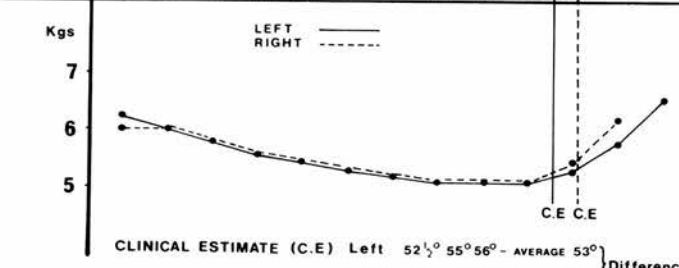
		35°	40°	45°	50°	55°	60°	65°	70°	75°	CLINICAL ESTIMATE (AVERAGE OF 3 MEASUREMENTS)
1.	LEFT	5.4	5.2	5.2	5.3	5.4	5.8	6.6	Kg		56° 57° 60° Av. 58°
	RIGHT	5.6	5.5	5.5	5.5	5.5	5.9	6.5	Kg		64° 63° 66° Av. 64°
2.	LEFT	4.3	4.7	4.8	4.8	5.0	5.4	6.0	6.9	7.8	65° 63° 66° Av. 65°
	RIGHT	4.2	4.3	4.5	4.6	4.6	4.9	5.3	5.8	6.8	65° 67° 68° Av. 67°
3.	LEFT	6.2	6.2	6.1	6.3	6.5	6.7				55° 56° 58° Av. 56°
	RIGHT	6.3	6.3	6.4	6.4	6.4	6.7	6.9			55° 54° 55° Av. 55°
4.	LEFT	5.8	5.6	6.0	5.6	6.5	7.8				52° 51° 50° Av. 51°
	RIGHT	4.8	4.8	4.6	5.0	5.1	5.7	6.4			54° 57° 60° Av. 57°
5.	LEFT	4.5	4.6	4.7	4.9	5.2	5.8				51° 55° 54° Av. 53°
	RIGHT	4.8	4.6	4.9	5.1	5.3	5.7				54° 52° 54° Av. 53°
6.	LEFT	4.2	4.1	4.1	4.2	4.5	4.9				54° 55° 56° Av. 55°
	RIGHT	4.1	3.9	3.9	4.0	4.2	4.5	5.1			53° 55° 57° Av. 55°
7.	LEFT	5.2	5.1	5.1	5.1	5.3	5.8	6.6			52½° 55° 56° Av. 53°
	RIGHT	5.2	5.1	5.1	5.1	5.4	6.2				56° 56½° 57° Av. 56°
8.	LEFT	6.1	6.2	6.8	7.4	8.0					45° 45° 43° Av. 44°
	RIGHT	5.6	5.5	6.0	6 4	7.4					48° 49° 50° Av. 49°
9.	LEFT	4.7	4.7	4.7	4.7	5.0	5.3	5.5	6.2		59° 61° 59° Av. 60°
	RIGHT	5.1	5.0	5.0	4.9	5.0	5.2	5.4	6.2		59° 65° 63° Av. 63°
10.	LEFT	6.1	6.5	6.9	7.4	8.1					43° 42° 42° Av. 42°
	RIGHT	5.9	6.2	6.6	7.1	7.6					46° 45° 46° Av. 46°

FIG. 6

7 MALE Aged 47

PERPENDICULAR TO HEEL - 140 cms
GREATER TROCHANTER TO HEEL - 93½ cms

	5°	10°	15°	20°	25°	30°	35°	40°	45°	50°	55°	60°	65°	
LEFT	6·2	6·0	5·8	5·6	5·5	5·3	5·2	5·1	5·1	5·1	5·3	5·8	6·6	Kgs
RIGHT	6·0	6·0	5·8	5·6	5·5	5·3	5·2	5·1	5·1	5·1	5·4	6·2		Kgs

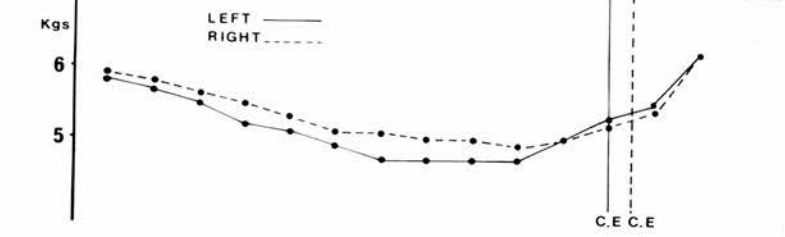


CLINICAL ESTIMATE (C.E) Left 52½° 55° 56° - AVERAGE 53° } Difference .. 3°
Right 56° 56½° 57° - AVERAGE 56° }

9 MALE Aged 37

PERPENDICULAR TO HEEL - 140 cms
GREATER TROCHANTER TO HEEL - 92 cms

	5°	10°	15°	20°	25°	30°	35°	40°	45°	50°	55°	60°	65°	70°	
LEFT	5·8	5·7	5·5	5·2	5·1	4·9	4·7	4·7	4·7	4·7	5·0	5·3	5·5	6·2	Kgs
RIGHT	5·9	5·8	5·6	5·5	5·3	5·1	5·1	5·0	5·0	4·9	5·0	5·2	5·4	6·2	Kgs

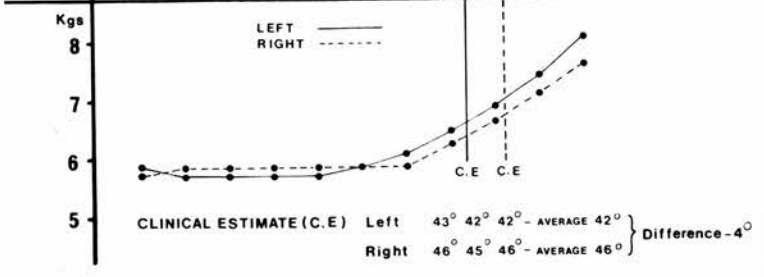


CLINICAL ESTIMATE (C.E) Left 59° 61° 59° - AVERAGE - 60° } Difference - 3°
Right 59° 65° 63° - AVERAGE - 63° }

(10) MALE Aged 22

PERPENDICULAR TO HEEL - 140 cms
GREATER TROCHANTER TO HEEL - 88 cms

	5°	10°	15°	20°	25°	30°	35°	40°	45°	50°	55°	60°	65°	70°	
LEFT	5·9	5·8	5·8	5·8	5·8	5·9	6·1	6·5	6·9	7·4	8·1				Kgs
RIGHT	5·8	5·9	5·9	5·9	5·9	5·9	5·9	6·2	6·6	7·1	7·6				Kgs



CLINICAL ESTIMATE (C.E) Left 43° 42° 42° - AVERAGE 42° } Difference - 4°
Right 46° 45° 46° - AVERAGE 46° }

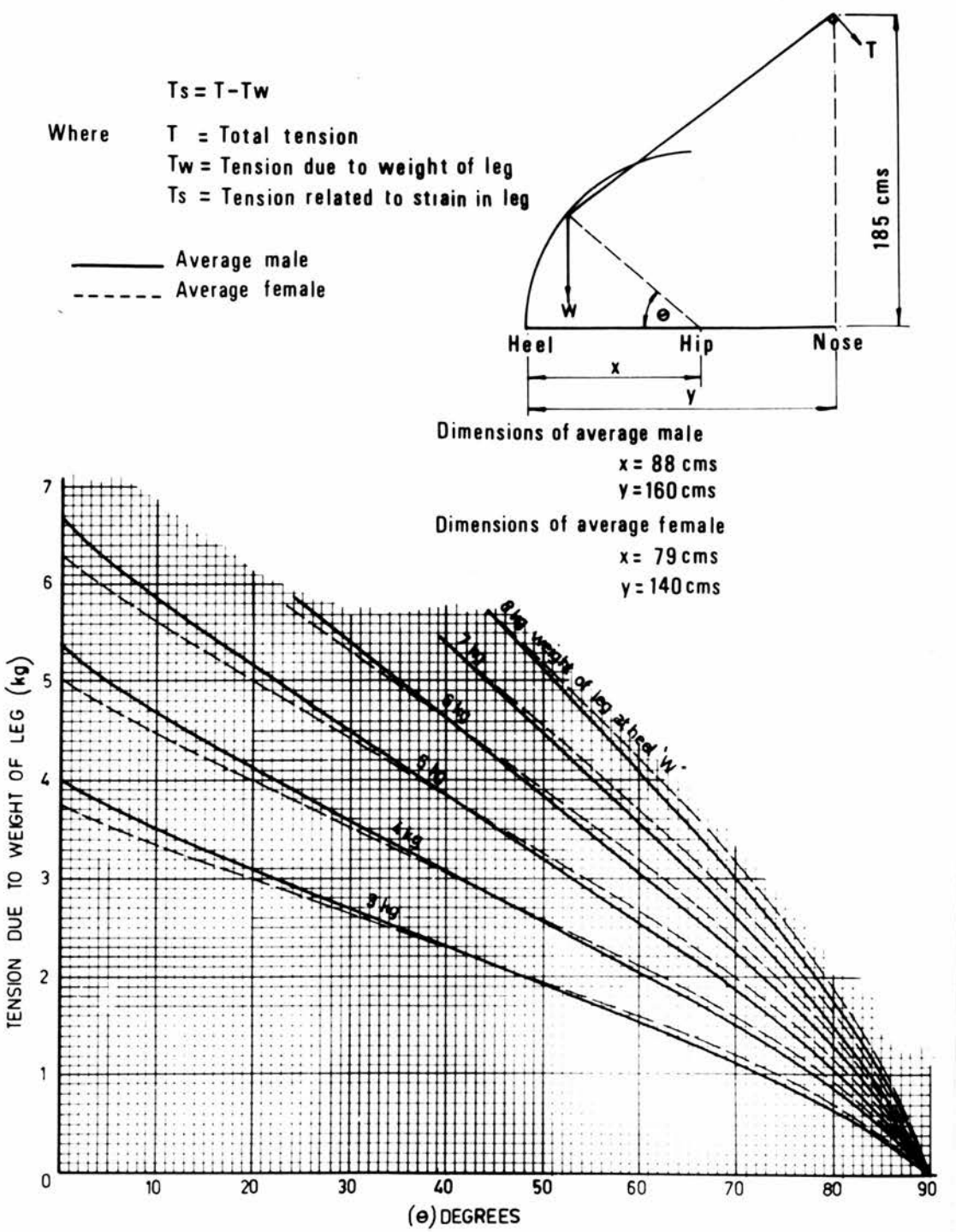
DISCUSSION

The maximum difference in the clinical estimate between the two sides was six degrees and the minimum was nil degrees.

In all the subjects there was a marked increase in the tension before the clinically determined end point. The clinical end point was at differing positions of the rising tension curve and where there was a difference of the clinical end point between the two sides this was not always reflected in the corresponding tension curves. Thus the tension measurement is more reliable and objective.

If a leg with a vertical weight at the heel varying from 3 to 8 Kgs. in the horizontal position is then lifted through an arc of 90 degrees and if there is no resistance to that movement the resultant tension curve from the subject positioned approximately as in this trial would be as shown in Fig. 7. The tension measurements in this trial are not like these curves because of the gradually increasing resistance to hip joint flexion. This resistance would be due to a gradual stretching of the skin, subcutaneous tissues, the adventitial tissue in and around the hip extensor muscles, and the inherent resistance of the muscles to stretch. The dramatic increase in tension is obviously indicative of the increasing resistance to deflexion of the lumbar spine

FIG. 7


Ts = T-Tw
Where T = Total tension
Tw = Tension due to weight of leg
Ts = Tension related to strain in leg
Average male
Average female
T
185 cms
W
θ
Heel
Hip
Nose
x
y
Dimensions of average male
x = 88 cms
y = 160 cms
Dimensions of average female
x = 79 cms
y = 140 cms
8 kg weight of leg at bed 'W'
7 kg
6 kg
5 kg
4 kg
3 kg
TENSION DUE TO WEIGHT OF LEG (kg)
(θ) DEGREES
0
1
2
3
4
5
6
7
10
20
30
40
50
60
70
80
90

and extension of the opposite hip, caused by the pelvic rotation which must take place when all the slack has been taken out of the hamstring muscles. This is confirmed by raising the leg with the knee flexed. No such rotation and tension change takes place.

The dramatic increase in tension and at what angle this takes place is thus an indication of the available passive stretch in mainly the hamstring group of muscles. This may be due to inherent length and or the amount of reflex muscle activity provoked by the stretching. The latter depends on the subject's ability to relax and control his own motor neurones. Basmajian (1976) showed that by consciously relaxing a muscle one can completely abolish neuromuscular activity. The different shapes of the tension curves in this study reflect a varying ability to relax.

The gradual stretching of the muscle would provoke afferent discharge from the muscle spindle. Leavitt et al (1964) showed that even quite rapid stretching of consciously relaxed muscles provoked no neuromuscular activity. This demonstrates the over-riding control of the higher centres on reflex motor neurone activity. The rate of stretch in this study was very slow and it should be well within the subject's ability to suppress reflex activity.

Stevens et al (1974) studied muscle activity in

the hamstring muscles of normal subjects whilst these were being passively stretched. They compared one group of subjects with tight hamstrings against a group with loose hamstrings. Those with tight muscles showed greater motor neurone activity during the stretch and this started 40 degrees short of the full stretch as compared with 20 degrees in the loose group. These studies were made with the pelvis and thigh fixed at about 60 degrees of flexion and the muscle activity measured whilst the knee was passively extended.

The candidate tested a small group of subjects in an attempt to correlate the amount of muscle activity in the hamstrings at or near the limit of passive stretch, using electromyographic recordings from the medial hamstrings. In two out of five subjects little if any extra activity was detected. The subjects used were well versed in the art of relaxation.

Thus it is evident that the available passive stretch is dependant mainly on the inherent length of the muscle but this may be influenced by reflex muscle contraction. But if, on the other hand, a patient with predominantly unilateral symptoms has evidence that the available stretch in the hamstrings on the affected side is markedly reduced, this should imply an increased excitability of the motor neurones on this side, either

directly or indirectly through increased activity of the gamma motor nerves to the muscle spindle.

ACKNOWLEDGEMENTS

The modified boot was constructed by Mr. P. Jarvie, Physiotherapist, Waikato Hospital. The candidate is also grateful for his invaluable help in organising teams of helpers, and for the many hours of his free time spent in assisting and advising.

Fig. 7 was produced for the candidate by Mr. J. Briggs, M.I.C.E., N.Z.I.E., Civil Engineer, Hamilton.

REFERENCES

Basmajian, J. V. (1976) Neurophysiologic Aspects of Rehabilitation Medicine. Ed. Buerger, A. A., and Tobis, J. S. p. 5 Illinois : Thomas.

Leavitt, L. A. Beasley, W. C. (1964) Clinical application of quantitative methods in the study of spasticity. Clin. Pharmacol. Ther. 5 : 918

Stevens, A., Stijns, H., Rosselle, N., Stappaerts, K., Michels, A. (1974) Slowly stretching the hamstrings and compliance. Preliminary report. Electromyog. Clin. Neurophysiol. 14 : 495 - 496

3. AN EVALUATION OF THE EFFECT OF MANIPULATION ON A PATIENT WITH UNILATERAL RESTRICTION OF PASSIVE HAMSTRING STRETCH

INTRODUCTION

Manipulative therapy has been with us for a very long time. In spite of this longevity there is still no evidence that manipulation results in measurable changes. Having used manipulation for a number of years, when it was felt to be indicated, the candidate still cannot predict with any degree of certainty whether it is going to be helpful or not. In the setting of general practice this is no real disadvantage as long as it is accepted as a trial of treatment.

Previous attempts at evaluating the effectiveness of manipulation have suffered from serious shortcomings. The two most quoted studies in the literature are those by Glover et al (1974) and Doran et al (1975). In the former the manipulative technique used would be considered ineffectual by those experienced in manipulation. In the latter a majority of the cases included in the trial would be considered by those experienced in this field as unlikely to benefit from manipulation. Furthermore, the techniques of manipulation were not standardised. The word manipulation can have a different meaning to different people.

To evaluate the effectiveness of any form of treatment in a problem such as back pain by comparing different groups of patients is virtually impossible. We as yet do not know what is causing the pain in the majority of patients. The main symptom is pain. There are few objective findings to measure, and sooner or later the majority of patients recover spontaneously, whatever the form of treatment or lack of it.

From the aspect of research, in order to cut down the innumerable variables involved in comparing different patients with a little understood pain, it would be convenient if one could compare the two sides of the same patient, in the assessment of the effectiveness of any treatment.

As previously noted, in a proportion of patients with predominantly one sided low back pain there is a clinically determined restriction of available passive stretch in the hamstring muscles on the painful side. It has also been observed that after manipulation this restriction disappears. Further studies using a more objective measurement have shown that this change can be demonstrated by measuring the varying tension as the legs are being raised, using the method already described.

It should therefore be possible to assess the

effect of treatment by comparing changes on the two sides of the same patient. This would eliminate the variables associated with comparisons between different individuals.

As an initial trial one such patient has been studied in detail.

CLINICAL DETAILS

Mr. R. A. D. Aged 31. Occupation: Shop manager.

History: Present attack

He noticed an ache in his right low back after a game of golf one month ago. This has persisted and has not improved. He is comfortable in bed and reasonably comfortable when he first gets up. The discomfort is aggravated by any bending, prolonged standing and sitting, particularly in a car.

Past attacks

He suffered his first attack of low back pain nine years ago as the result of a fall. This gradually settled with no specific treatment. He was seen by the candidate seventeen months ago with a history of having slipped and fallen whilst carrying a heavy weight one year prior to this. He had a persistent right low back ache since then.

This ache went after three rotational manipulations of the right lumbo-sacral region of his spine. It was noted at that time that he had tight hamstring muscles and he stated that he has always had difficulty in touching his toes. He was instructed in hamstring stretch exercises. He kept these up for three months and then forgot about them/

Examination:

Tall. Average build.

Standing When bending forward, keeping his knees straight, in an attempt to touch his toes, palpation of the posterior superior iliac spines showed that the right side was held back.

Sitting The Posterior Superior Iliac Spines moved evenly when bending forward to put his head between his knees.

Lying Supine: The passive hamstring stretch test showed that both muscles were tight and the right one was tighter than the left.

Prone: There was tenderness to pressure which was maximal between the fifth lumbar and first sacral spinous processes. The paravertebral muscles at this level were more tense on the right side. He had a typical trigger point on his right gluteal muscles.

METHOD

First, the clinical end point of the passive hamstring stretch test was assessed. The tension measurements were then taken, as previously described.

Next, the left side of the patient's lower lumbar spine was manipulated, using one modification of what the osteopaths term "the million dollar roll". (see Fisk 1977, also Appendix). This ensured that the facet joints on the left side were gapped at the desired level.

Both the clinical end points and the tension measurements were then re-checked.

Next, the right side of the patient's lower lumbar spine was manipulated, again ensuring that the right lumbo-sacral facet joint was gapped.

Again, the clinical end point of the passive hamstring stretch and the tension measurements were re-checked.

All the estimates of the clinical end points and the manipulations were done by the candidate, otherwise he took no further active part in the measurements. Each set of recordings were made on separate sheets of paper, and the assistant making the first recordings did not know on which side the patient had pain. The following

recordings were taken by another assistant who was also unaware of where the patient had pain and also did not know which side of the patient had been manipulated. Furthermore, the patient was unaware of what was being recorded and what the expected results should be.

The measurements were checked the next day and then six days later. At this time he was manipulated again on the right side only and re-checked. He was again re-checked next day and again six days later.

RESULTS

See Fig. 8.

The tension is measured in kilograms.

The main features of this table are clearer when illustrated as a graph. See Fig. 9.

DISCUSSION

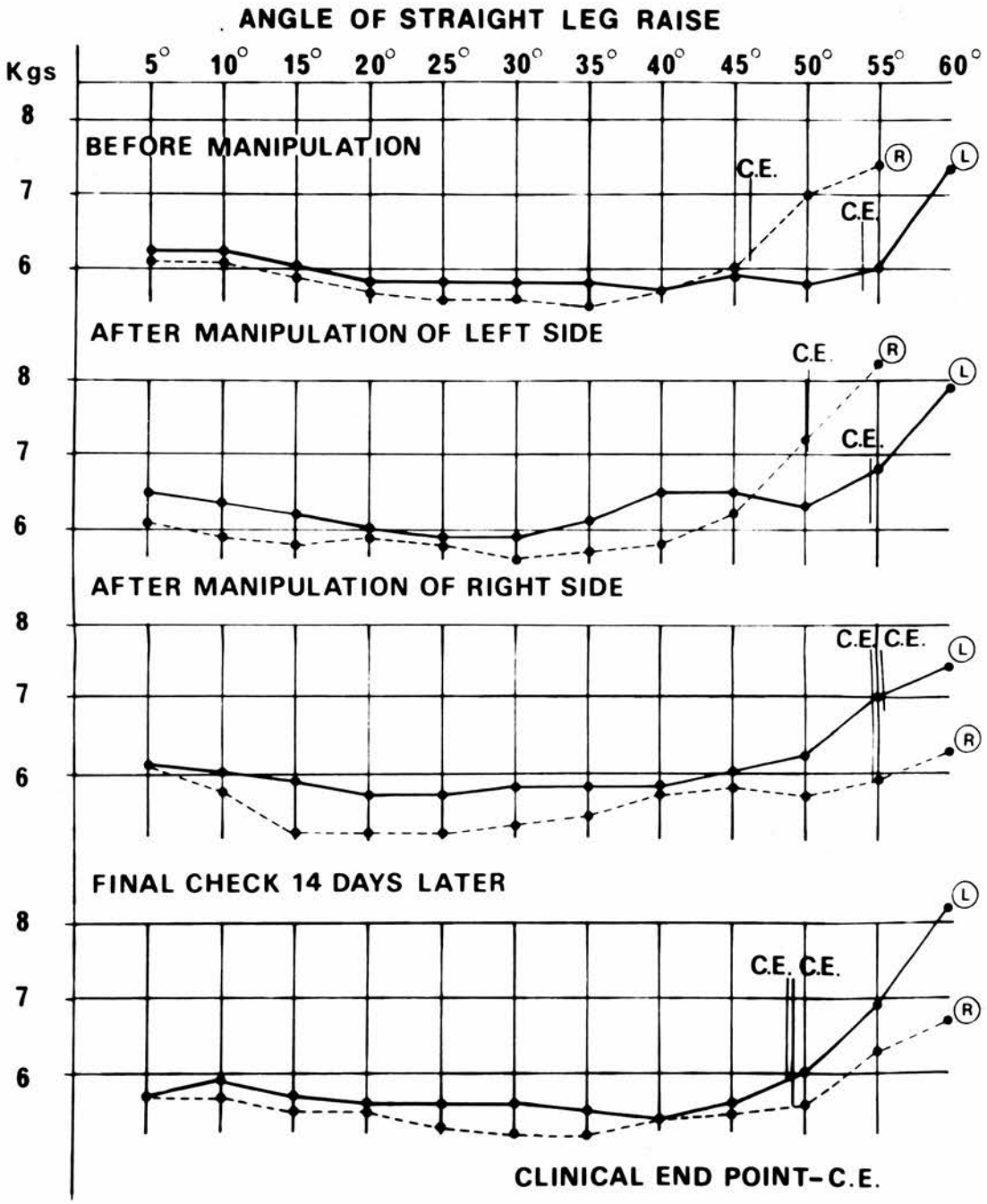
The pain free side of the patient was deliberately manipulated first. This was in order to determine how important the gapping phenomena was in the manipulative technique used, rather than just the stretching of the paravertebral structures.

The tension measurements were first taken on the non manipulated side, the clinical measurements having been taken prior to this. It has been found that even in normal subjects there is a short lasting increase in the passive hamstring stretch after manipulation. The length

FIG 8

	25°	30°	35°	40°	45°	50°	55°	60°	65°	CLINICAL ESTIMATE		
LEFT	5.8	5.8	5.8	5.7	5.9	5.8	6.0	7.3		54°	BEFORE MANIPULATION	
RIGHT	5.6	5.6	5.5	5.7	6.0	6.9	7.4			46°		
LEFT	5.9	5.9	6.1	6.5	6.5	6.3	6.8	7.8	8.4	54½°	AFTER LEFT MANIPULATION	
RIGHT	5.8	5.6	5.7	5.8	6.2	7.2	8.2			50°		
LEFT	5.7	5.8	5.8	5.8	6.0	6.2	7.0	7.4	8.6	55°	AFTER RIGHT MANIPULATION	SYMPTOMS
RIGHT	5.2	5.3	5.4	5.7	5.8	5.7	5.9	6.3	7.2	55°		FEELS EASIER
LEFT	5.9	5.8	5.9	5.8	6.0	6.8	7.2	8.0		46°	NEXT DAY	FEELS FREER BUT STILL PAINFUL ON RIGHT SIDE
RIGHT	6.2	6.1	6.0	6.4	7.0	8.0				43°		
LEFT	5.5	5.5	5.7	5.8	5.8	6.3	7.4			49°	6 DAYS LATER BEFORE MANIPULATION	STILL STIFF & SORE AFTER 3 DAYS SITTING IN A CONFERENCE
RIGHT	6.0	5.6	5.8	6.1	6.8	8.0				40°		
LEFT	6.0	5.9	6.0	6.0	6.6	7.2	8.2			49°	AFTER RIGHT MANIPULATION	MUCH MORE COMFORTABLE
RIGHT	5.8	5.3	5.6	5.8	5.9	5.9	6.4	7.4	8.0	53°		
LEFT	6.0	5.9	5.9	6.0	6.0	6.6	7.4			42°	NEXT DAY	FEELS FREER LESS PAIN
RIGHT	6.0	6.0	5.8	5.6	5.8	6.0	7.0			42°		
LEFT	5.6	5.6	5.5	5.4	5.6	6.0	6.9	8.2		48°	6 DAYS LATER	NO SYMPTOMS
RIGHT	5.3	5.2	5.2	5.4	5.5	5.6	6.3	6.7	7.6	48°		

FIG. 9


ANGLE OF STRAIGHT LEG RAISE
Kgs
5° 10° 15° 20° 25° 30° 35° 40° 45° 50° 55° 60°
8
7
6
BEFORE MANIPULATION
C.E.
C.E.
R
L
AFTER MANIPULATION OF LEFT SIDE
C.E.
C.E.
AFTER MANIPULATION OF RIGHT SIDE
C.E. C.E.
FINAL CHECK 14 DAYS LATER
C.E. C.E.
CLINICAL END POINT- C.E.

of this reflex change has not been measured but it probably less than a minute.

These results objectively demonstrate an alteration in the resistance to hip flexion on the affected side. This alteration was most marked after gapping the lumbo-sacral joint on the painful side. Thus, the cavitation phenomena rather than just stretching would appear to be important. As has been described before (Fisk, 1977) in the technique used the facet joints to be manipulated have their two surfaces in full apposition to each other. That is, in the neutral position, neither flexed or extended. In Glover's series the gapping effect was noted in only 70% of those treated by manipulation, and judging by the technique used this probably occurred at a higher level. It is easier to gap normal joints rather than those that are under increased tension. This may be one of the reasons for his inconclusive results.

The only possible explanation that the candidate can think of that could explain this sudden change in tension is a reflex alteration in the excitability of the motor neurones supplying the hamstring muscles. It has been suggested that there may be a freeing of the sacro-iliac joint on this side, but clinical observation shows the opposite to be so. As already mentioned the posterior superior iliac spine is held back on the affected side as the patient bends forward whilst attempting to touch the toes, with

the knees straight, but not so when bending forward from the sitting position. Furthermore they move synchronously after manipulation.

In the technique used there is no direct stretching or pulling on the hamstring muscles therefore the effect of the manipulation is likely to be at the spinal level.

The reflex excitability of the motor neurones would seem to have a segmental distribution. Hence the focal paravertebral muscle tension, the trigger point in the gluteal muscles and the hamstring tightness. The involved muscles derive their nerve supply from mainly the L.5, S.1 segments. It is also possible to clinically determine a reduction in the passive stretch of the gluteal muscles. It may be possible to further demonstrate this segmental excitation by showing that stretching of the gastro-soleus muscles influences the passive stretch in the hamstrings. These muscles also derive their nerve supply from the same sggments.

The possible importance of disordered muscle activity in the pathogenesis of back pain has already been discussed (Fisk 1977). It is proposed that manipulation could alter this reflex excitability by stimulating powerful motor neurone inhibitors. These are probably the type $\overline{111}$ mechanoreceptors present in the intrinsic and

extrinsic facet joint ligaments (Wyke 1972), which are identical to Golgi tendon organs. It would also seem logical that this reflex excitability that is alterable is brought about by increased activity in the gamma motor nerves. The smaller gamma nerves would be more vulnerable to reflex influences. This may also explain why claims for different methods of treatment, such as different techniques of manipulation and different ways of treating trigger points, are probably all equally successful or equally unsuccessful.

Before further discussion takes place it is important to confirm these initial findings.

REFERENCES

Doran, D. M. L., Newell, D. J. (1975)
Manipulation in treatment of low back pain : a multicentre study.
B.M.J. 2 : 161 - 164

Fisk, J. W. (1977)
A Practical Guide to Management of the Painful Neck and Back.
p. 91, 141 - 147.
Springfield : Thomas.

Glover, J. R., Morris, J. G., Khosla, T. (1974)
Back pain : a randomised clinical trial of rotational manipulation of the trunk.
Brit. J. Indust. Med. 31 : 59 - 64

Wyke, B. (1972)
Articular neurology : A review.
Physiotherapy. 58 : 94 - 99

4. A CONTROLLED TRIAL OF MANIPULATION IN A SELECTED GROUP OF PATIENTS WITH LOW BACK PAIN FAVOURING ONE SIDE.

INTRODUCTION

The initial trial with one patient complaining of one sided low back pain showed that the painful side had a clinically estimated reduction in the available stretch in the hamstring muscles. This was reflected by a tension curve that increased at a lower angle on the painful side. Furthermore, this was altered by manipulation of the painful side. A follow-up study of this patient showed that alteration in the tension measurements, comparing one side against the other, reflected clinical pregress.

It was felt that these findings probably reflected changes in the reflex excitability of the motor neurones supplying the hamstring muscles. The object of this study is to further explore this hypothesis and possibly to demonstrate a measurable effect of manipulation on carefully selected patients.

METHOD

Ten carefully selected patients, mainly from the candidate's own practice, with a particular pattern of

clinical findings, were asked to take part in this trial. They all had in common low back pain favouring one side of the back. They had the following characteristics:

See Fig. 10.

On examination, all ten had reduction in the passive hamstring stretch on the painful side, trigger points in the gluteal muscles and focal increased para-vertebral muscle tension on the painful side. The posterior superior iliac spine on the painful side was held back on forward bending from the standing position in six patients. One was too fat for satisfactory examination. In eight patients tenderness to palpation was maximal between the fifth lumbar and first sacral spines, between the fourth and fifth lumbar in one, and both were equally tender in one.

The tension in each leg was measured at five degree increments as described before. The non-painful side was then manipulated using one specific technique (see Appendix)., and the measurements repeated. Next, the painful side was manipulated and the measurements again repeated. To ensure maximum objectivity the observer taking the first tension measurements did not know which side was painful. Later measurements were taken by another observer who also did not know which side was painful, or which side had been manipulated. Each set of recordings

FIG 10

		PATIENTS									
		1	2	3	4	5	6	7	8	9	10
AGE		31	31	40	39	39	45	55	25	37	29
SEX		M	F	M	M	M	F	M	M	F	M
DURATION OF SYMPTOMS (DAYS)		28	8	21	5	35	2	6	17	21	2
GROUP (SEE BELOW)		C	C	C	D	D	E	A	C	C	B
PAINFUL SIDE		R	L	L	R	L	L	R	R	R	L
AGGRAVATED BY: -	BENDING	x	x	x	x	x	x	x	x	x	x
	SITTING	x	x	x	x	x		x	x	x	x
	STANDING	x		x				x			
	WALKING			x				x			
	GETTING OUT OF BED		x		x	x		x	x	x	
	REST IN BED			x							x
RELIEVED BY: -	REST	x	x		x	x	x	x	x	x	
	WALKING		x		x	x				x	
	STANDING						x			x	
PAST ATTACKS M - MANY F - FEW		2	M	M	M	1	M	M	M	F	M
YEARS SINCE FIRST ATTACK		9	9	25	20	3	4	13	6	3	12
NUMBER OF MANIPULATIONS		2	1	1	1	1	1	1	1	2	1
? OTHER TREATMENT		No	No	No	No	No	No	No	No	Yes	No

GROUPS: A - Severe strain, gradual onset of pain

B - Severe strain, sudden onset of pain

C - Protracted strain

D - Mild strain, sudden onset of pain

E - No strain

(See Fisk 1977 (A))

OTHER TREATMENT (9) One week of ultrasonics and pressuring to the trigger point in the gluteals.

was made on a separate sheet of paper. The candidate took no part in the proceedings except to do the manipulations and pull on the rope that elevated the leg.

Each patient was followed up until he or she was free of symptoms. Repeat tension measurements were taken at these times and if it was felt that further manipulation was indicated measurements were again taken before and after.

Whilst collecting this series of cases ten rather unwilling subjects, most of them being members of the resident staff of the Waikato Hospital, who were free of back pain, were used as controls. Each of them went through the same procedure. Tension measurements were taken before manipulation, after manipulation of one side and again after manipulation of the other. The observer taking the measurements did not know which side, if any, had been manipulated.

The controls were aged between 22 and 47 years. There were nine males and one female.

The time of the manipulation was recorded on a stop watch. Unbeknown to the observers, mainly members of the physiotherapy department, the first tension measurements were taken from the side not manipulated and the time of the last tension measurement after the

manipulation was recorded. The minimum time of this recording was four minutes.

The Salter tension gauge used for these measurements was checked by the hospital engineers. Between three and fifteen kilograms, at one kilogram intervals, it was found to be completely accurate.

The observers recording the measurements were first carefully instructed on how to read the scale and sample recordings were checked by others. The Salter scale was marked at 0.2 Kg. intervals. When the scale pointer fell between these markings the result was recorded as the odd number. It would therefore seem to be a reasonable estimate that the limits of accuracy of the readings would be ± 0.05 Kgs.

RESULTS

The change in the tension curves is best illustrated by taking the last two readings that were complete for both the left and right leg throughout the series of measurements:

See Figs. 11 and 12

(For complete measurements, see Appendix).

The alteration in the difference in tension between the two sides after manipulation of the patients

FIG 11

NORMAL CONTROLS. THE LAST TWO READINGS IN BOTH LEGS, BEFORE AND AFTER MANIPULATION (Kgs)

(THE INITIALLY TIGHTER SIDE IS UNDERLINED)

	BEFORE MANIPULATION		AFTER MANIPULATION OF FIRST SIDE		AFTER MANIPULATION OF BOTH SIDES	
1	55°	60°	R 55°	60°	55°	60°
LEFT	6.0	6.5	6.1	6.6	6.0	6.4
RIGHT	6.2	6.7	6.0	6.4	6.1	6.7
2	55°	60°	L 55°	60°	55°	60°
LEFT	7.0	7.5	6.7	7.1	6.9	7.4
RIGHT	6.9	7.1	6.6	7.0	6.7	7.1
3	55°	60°	L 55°	60°	55°	60°
LEFT	5.6	5.9	5.6	5.9	5.4	5.8
RIGHT	5.5	5.7	5.5	5.8	5.4	5.7
4	45°	50°	L 45°	50°	45°	50°
LEFT	7.9	8.2	7.6	7.8	7.9	8.2
RIGHT	8.0	8.4	7.4	7.6	7.9	8.4
5	55°	60°	L 55°	60°	55°	60°
LEFT	5.3	5.8	5.3	6.0	5.4	5.7
RIGHT	5.4	6.2	5.6	6.4	5.6	6.2
6	60°	65°	L 60°	65°	60°	65°
LEFT	5.5	6.0	5.5	6.4 5.	5.7	6.4
RIGHT	5.6	6.2	5.8	6.1	5.7	6.1
7	50°	55°	L 50°	55°	50°	55°
LEFT	6.4	6.9	6.4	6.7	6.4	6.9
RIGHT	6.5	6.9	6.2	6.6	6.3	6.7
8	60°	65°	R 60°	65°	60°	65°
LEFT	6.7	7.2	6.9	7.4	6.8	7.5
RIGHT	6.2	6.8	6.1	6.9	6.2	6.7
9	60°	65°	R 60°	65°	60°	65°
LEFT	6.0	6.3	5.8	6.3	7.0	7.6
RIGHT	5.7	6.0	6.0	6.4	6.0	6.7
10	65°	70°	L 65°	70°	65°	70°
LEFT	5.9	6.0	5.7	6.1	5.7	6.2
RIGHT	5.3	5.9	5.9	6.5	5.6	6.0

FIG 12

TENSION MEASUREMENTS. LAST TWO READINGS IN BOTH LEGS, BEFORE AND AFTER MANIPULATION (Kgs)

	BEFORE MANIPULATION		AFTER MANIPULATION OF "WRONG" SIDE		AFTER MANIPULATION OF PAINFUL SIDE		FINAL CHECK WHEN FREE OF PAIN		
1	50°	55°	50°	55°	50°	55°	50°	55°	)
LEFT	5.8	6.0	6.3	6.8	6.2	7.0	6.0	6.9	) 14 days later
RIGHT	6.9	7.4	7.2	8.2	5.7	5.9	5.6	6.3	
2	60°	65°	60°	65°	60°	65°	60°	65°	)
LEFT	4.5	5.0	4.3	5.0	4.0	4.4	3.8	4.8	) 5 days later
RIGHT	4.0	4.2	3.8	4.2	4.0	4.4	4.0	4.8	
3	65°	70°			65°	70°	65°	70°	
LEFT	6.0	7.0			5.5	5.9	6.0	6.2	) 2 days later
RIGHT	5.7	6.0			5.9	6.2	7.4	8.0	
4	70°	75°	70°	75°	70°	75°	70°	75°	)
LEFT	6.4	7.2	7.0	7.8	7.0	8.2	6.5	7.0	) 3 days later
RIGHT	7.2	7.8	7.1	8.0	7.1	8.1	6.3	6.9	
5	65°	70°	65°	70°	65°	70°	65°	70°	)
LEFT	7.6	8.1	7.7	8.2	7.3	7.9	7.8	8.3	) 3 days later
RIGHT	7.4	7.9	7.1	7.5	7.5	8.2	8.2	9.1	
6	60°	65°	60°	65°	60°	65°	60°	65°	
LEFT	5.0	5.6	4.4	4.8	4.2	4.6	4.5	5.2	)
RIGHT	4.5	4.7	4.3	4.5	4.1	4.4	4.2	4.5	) 3 days later
7	65°	70°			65°	70°	65°	70°	
LEFT	5.4	5.8			5.6	6.0	5.5	6.1	)
RIGHT	6.2	7.3			5.1	5.3	5.1	5.5	) 3 days later
8	55°	60°	55°	60°	55°	60°	55°	60°	
LEFT	6.4	7.1	6.9	7.4	7.2	8.1	6.8	7.4	)
RIGHT	6.8	7.5	7.9	8.4	6.7	7.4	6.7	7.0	) 5 days later
9	70°	75°	70°	75°	70°	75°	70°	75°	
LEFT	4.9	6.0	5.0	5.6	4.9	5.8	5.1	NOT	)
RIGHT	4.2	4.8	4.3	4.9	4.7	5.4	5.1	MEASURED	) 18 days later
10	55°	60°	55°	60°	55°	60°	55°	60°	
LEFT	7.6	8.2	7.3	8.1	7.8	8.3	7.3	7.8	)
RIGHT	7.0	7.6	7.1	7.3	7.7	8.5	7.4	8.1	) 7 days later

N. B. THE PAINFUL SIDE IS UNDERLINED

illustrates any change that has been effected by treatment. This is denoted as the swing of the painful side towards the non painful side, taking the latter as a zero reading. Likewise, in the controls the swing of the tighter side towards the less tight side is taken as a positive swing. For statistical analysis each of the last two readings are pooled, averaged, and compared.

See Figs. 13 & 14.

STATISTICAL ANALYSIS

For the ten cases the load before treatment measured over the last two recorded values was differenced between the affected leg and the unaffected leg. The test was repeated after manipulation and the change evaluated. This change represents the reduction in load due to treatment and the individuals' values are presented in Figs. 13 and 14.

The same measurements were made on ten normals, the change being expressed as a reduction in load in the leg showing the higher load before treatment minus the reduction in the leg showing the lower load before treatment. This standardised the comparison of cases versus normals.

See Fig. 15

FIG 13

DIFFERENCES IN TENSION BETWEEN THE TWO SIDES BEFORE AND AFTER MANIPULATION (Kgs).

		BEFORE MANIPULATION			AFTER MANIPULATION OF FIRST SIDE				AFTER MANIPULATION OF SECOND SIDE			
		1	2	AVERAGE	1	2	AVERAGE	SWING	1	2	AVERAGE	SWING
CONTROLS	1	0.2	0.2	0.2	R -0.1	-0.2	-0.15	0.35	0.1	0.3	0.20	0.00
	2	0.1	0.4	0.25	L 0.1	0.1	0.1	0.15	0.2	0.3	0.25	0.00
	3	0.1	0.2	0.15	L 0.1	0.1	0.1	0.05	0.0	0.1	0.05	0.10
	4	0.1	0.2	0.15	L-0.2	-0.2	0.2	0.35	0.0	0.2	0.10	0.05
	5	0.1	0.4	0.25	L 0.3	0.4	0.35	-0.1	0.2	0.5	0.35	-0.1
	6	0.1	0.2	0.15	L 0.3	0.3	-0.3	-0.15	0.0	-0.3	-0.15	0.3
	7	0.1	0.0	0.05	L-0.2	-0.1	-0.15	0.2	-0.1	-0.2	-0.15	0.2
	8	0.5	0.4	0.45	R 0.8	0.5	-0.65	-0.2	0.6	0.8	0.7	-0.25
	9	0.3	0.3	0.3	R 0.2	0.1	0.15	0.15	-1.0	-0.95	-0.65	
	10	0.6	0.1	0.35	R-0.2	-0.4	-0.3	0.65	0.1	0.2	0.15	0.2

FIG 14

DIFFERENCES IN TENSION BETWEEN THE PAINFUL AND NON PAINFUL SIDES OF THE LAST 2 READINGS BEFORE AND AFTER MANIPULATION (Kgs).

		BEFORE MANIPULATION			AFTER MANIPULATION OF NON-PAINFUL SIDE				AFTER MANIPULATION OF PAINFUL SIDE			
		1	2	AVERAGE	1	2	AVERAGE	SWING	1	2	AVERAGE	SWING
PATIENTS	1	1.1	1.4	1.25	0.9	1.4	1.15	0.10	-0.5	-1.1	-0.8	2.05
	2	0.5	0.8	0.65	0.5	0.8	0.65	0.00	0.0	0.0	0.0	0.65
	3	0.3	1.0	0.65	FAILED TO MANIPULATE THIS SIDE				-0.4	-0.3	-0.35	1.00
	4	0.8	0.6	0.7	0.1	0.2	0.15	0.55	0.0	0.0	0.0	0.65
	5	0.2	0.2	0.2	0.6	0.7	0.65	-0.45	-0.2	-0.3	-0.25	0.45
	6	0.5	0.9	0.7	0.1	0.3	0.2	0.5	0.1	0.2	0.15	0.55
	7	0.8	1.5	1.15	FAILED TO MANIPULATE THIS SIDE				-0.5	-0.7	-0.6	1.75
	8	0.4	0.4	0.4	1.0	1.0	1.0	-0.6	-0.5	-0.7	-0.6	1.0
	9	0.7	1.2	0.95	0.7	1.7	1.2	-0.25	0.2	0.4	0.3	0.65
	10	0.6	0.6	0.6	0.2	0.8	0.5	0.1	0.1	-0.2	-0.05	0.65

N.B. A NEGATIVE SIGN IN THE SWING MEANS A FURTHER SWING OF THE TIGHTER SIDE AWAY FROM THE LESS TIGHT SIDE

FIG 15

MEAN REDUCTION IN LOAD DUE TO TREATMENT (Kgs).

	CASES	NORMALS
	2.05	0.00
	0.65	0.00
	1.00	0.10
	0.70	0.05
	0.45	-0.10
	0.55	0.3
	1.75	0.2
	1.00	-0.25
	0.65	-0.65
	0.65	0.2
MEAN	0.945	-0.015
STANDARD DEVIATION	0.537	0.274

FIG 16

PERPENDICULAR TO HEEL - 140 cm
GREATER TROCHANTER TO HEEL - 97 cm

3 READINGS TAKEN AT ½ HOUR INTERVALS

	LEFT			RIGHT		
	1	2	3	1	2	3
5°	6.0	6.0	5.9	5.9	6.1	6.1
10°	5.9	5.9	5.8	5.9	6.0	6.0
15°	5.7	5.8	5.7	5.8	5.8	6.0
20°	5.5	5.6	5.6	5.5	5.7	5.8
25°	5.5	5.6	5.5	5.4	5.7	5.7
30°	5.3	5.3	5.4	5.4	5.5	5.6
35°	5.2	5.3	5.3	5.3	5.5	5.5
40°	5.1	5.2	5.3	5.3	5.5	5.5
45°	5.1	5.2	5.3	5.3	5.6	5.6
50°	5.1	5.4	5.3	5.3	5.7	5.8
55°	5.5	5.7	5.5	5.7	5.9	6.1
60°	6.0	6.1	6.1	6.3	6.3	6.4
65°	6.8	6.9	6.7	7.1	7.1	7.1
70°						

MEASUREMENTS TAKEN IN KILOGRAMS

The standard error of the difference between means was therefore 0.190 giving a t-value of 5.05, significant at the 0.1% level, ($P < 0.001$), using a two-tailed test for differing within-group variances (Fisher). The group variances differ at the 5% level of significance. If it is accepted that the treatment effect on normals has a mean value of zero in a very large population then the difference between cases and normals yielded an even stronger result.

DISCUSSION

Thus we have objective statistically valid evidence that manipulation has an effect on carefully selected patients. These patients demonstrate a difference in hamstring tension for a given arc of elevation of the two legs. This difference of tension is not present in controls, and that the relief of symptoms following manipulation is accompanied by the disappearance of this tension difference. In the light of present knowledge the understanding of this effect may only be speculative.

The patients selected for this trial all roughly approximate to the profile described before. It is of interest to note that 50% suffered no specific strain (Group C). This is a much higher percentage of the total than was found in a previous survey (Fisk 1977 (A)). A high proportion had suffered many previous attacks for up to 25 years. They did not follow the pattern of rapidly

degenerating disc disease, when the attacks tend to get more frequent and more severe. From personal experience of both hospital and general practice, this type of patient is rarely seen in the hospital outpatients. The degree of incapacity is usually not severe and persistent enough. Such patients often bypass their own practitioners, experience having taught them that rapid relief will be forthcoming from non-medical manipulators.

The 'wrong' side was deliberately manipulated first, in order to assess the importance of the gapping phenomena in affecting the tension change. Also, all of the patients had experienced manipulation before but were not knowledgeable enough to appreciate which was the right and which was the wrong side. In all but two cases where both sides were manipulated successfully the marked change in tension was not evident until after manipulation of the painful side (see Fig. 12). Thus, using the specified technique, the stretching of the paravertebral structures alone is usually not enough to effect the change. The technique also does not place any stretch on the hip extensor muscles.

It would seem unlikely that a healthy spinal joint complex could be injured to the extent of leading to long term discomfort after the often trivial strains as is so often the case. There is no doubt that the human disc is particularly prone to premature breakdown or

aging. This could lead to many potential pain provoking mechanisms, both discogenic and indirectly from other parts of the spinal joint complex. These could be due to the abnormal stresses and movement patterns resulting from the functional failure of the disc.

Possible pain provoking mechanisms in the spine have been the subject of an overwhelming mass of research literature and speculation over the past few years. There is a wide spectrum of treatment approaches, with so many undeniable cures and so many undeniable failures in patients with identical symptoms. The fact that there are advocates of so many treatment approaches leads one to speculate upon the whole symptom complex of low back pain, and as has been suggested by Rose (1975) it may well be determined ultimately that there is a common basis to all such procedures. The candidate's own such research suggests that this common basis is likely to be the correction of disordered muscle activity. (Fisk 1977 (B)).

All the trial patients had so-called myofascial trigger points. The aetiology of these is controversial. This has already been discussed. There is no doubt about their existence as a clinical entity but so far research has failed to demonstrate convincing evidence of increased focal muscle activity. Further research is in progress (Basmajian 1976). Perhaps the tender knot of palpable muscle is provoked by the examination. It is known that some skin afferent nerves are capable of

provoking increased gamma activity (Hunt 1951). It is likely that local treatment to these areas in the trial patients would most probably result in relief of the patient's symptoms. Case 9 was one such instance. After two manipulations she was not completely free of symptoms and the passive hamstring stretch was still reduced on the painful side. She had one week's local treatment to the gluteal trigger point, in the form of ultrasonics and deep pressuring, before she was completely free of symptoms. Methods of treating these trigger points have various advocates. It would seem likely that the various therapies are all equally successful or equally unsuccessful. It is also common experience that these trigger points may disappear after manipulation of the appropriate spinal segment, which also has local signs of over-stress. Also, trigger points are often found in the gluteal muscles in the presence of a known disc protrusion, and these do not go away whatever local treatment is used.

It would seem likely that these trigger points are part of a segmental increased excitability of motor neurones. This may start originally from stimulation of the pain receptors following some original strain, either in the spinal joint capsule or its ligaments, or, less likely, from some local muscle strain. Having once been produced why do they then often become self perpetuating after the initial strain has had adequate time to heal? It is known that the nociceptor nerves have a

polysynaptic connection with the alpha motor neurones (Wyke 1976). There is also an increase in gamma discharge on nociceptor stimulation. Granit (1975) called this a nociceptor reflex partnership. This is the obvious reflex pathway of protective muscle spasm. It is also possible that if this protective muscle spasm or increased tonus is prolonged the nociceptor nerve endings within the muscle could be depolarised either by chemicals released from the anoxic muscle or by mechanical distortion (Wyke 1976). This would create a vicious circle.

It is thought that the inflow from the nociceptor nerves may be effectively blocked by stimulation of high threshold large fibred mechanoreceptors, found in skin, muscles, tendons and the intrinsic and extrinsic ligaments of the synovial joints, by means of a collateral inhibitory mechanism, thus breaking into the vicious circle. This is the likely mechanism involved in such procedures as electrical skin stimulation, acupuncture, cold spray and stretching, etc.

Travell (1952) made the important observation that when the overlying skin is sprayed with ethyl chloride it should not be chilled too much. The likely reason for this is that light cooling leads to stimulation of skin proprioceptors that have a reflex inhibitory effect on the motor neurones, whereas lowering the temperature too much leads to reflex excitation of the motor neurones.

But this does not seem to be the complete answer. The patients involved in this trial were not suffering continuous discomfort. For instance, nearly all of them were quite comfortable resting in bed, and sometimes only certain movements provoked the discomfort.

It would seem reasonable to suppose that the maintenance of this vicious circle requires a further input. This could be supplied by increased activity of the small fibred joint proprioceptors. This activity could be provoked by the surrounding increased muscle tension.

The research by Denslow et al has already been discussed (see Survey of Previous Work). They showed that even in pain free subjects there was a fluctuating degree of motor neurone excitability from one segmental level to the next, and also from side to side, in the dorsal spine. Unlike the cervical and lumbar spine the nerve supply to the spinal joints, muscles, and skin in the dorsal region is confined to a strictly segmental level (Wyke 1970). It would seem obvious that this difference could only be manifested at the spinal reflex level.

As already mentioned, the only constantly discharging afferent joint receptors in the resting posterior spinal joints are the small fibred type 1 static and dynamic mechanoreceptors (Wyke 1967). Likewise the small

fibred gamma motor nerves to the muscle spindle are also constantly discharging. It is likely that these small fibred receptors have a polysynaptic connection to the gamma motor nerves rather than to the alpha motor nerves (Wyke 1967). It is also thought that for the localisation of deep pain input from other mechanoreceptors than just nociceptors is necessary (Wyke 1976). Again, the most likely source would be these type 1 receptors. It requires no great stretch of the imagination to postulate that individual posterior spinal joints could have varying activity in these receptors that could lead to varying excitability of the segmentally related motor neurones, via the gamma loop. It would only require minor changes in the capsule and attached ligaments to effect this change.

It would also seem reasonable to suggest that the act of gapping the posterior spinal joints would stimulate the large fibred high threshold type 3 joint receptors. These have a strong inhibitory influence on the segmentally related alpha motor neurones. Again, this effect would most likely be mediated by influence on the smaller fibred gamma nerves. This could thus be the mechanism whereby manipulation alters the 'gain' in the gamma loop, which leads to the segmentally related motor neurone excitability being turned down. Refined electrophysiological research would be necessary to test this hypothesis.

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REFERENCES

Basmajian, J. B. (1976) Electromyographic investigation of spasticity and muscle spasm. Physiotherapy. 62 : 319 - 323

Denslow, J. S., Korr, I. M., Krems, A. D. (1947) Quantitative studies of chronic facilitation in human motoneuron pools. Am. J. Physiol. 150 : 229 - 238.

Fisk, J. W. (1977 A) Approaches to the Validation of Manipulation Therapy. Ed. Buerger, A. A. & Tobis, J. S. 236 - 270. Springfield : Thomas.

Fisk, J. W. (1977 B) A Practical Guide to Management of the Painful Neck and Back. 94 - 101. Springfield : Thomas

Granit, R. (1975) The functional role of the muscle spindles : facts and hypotheses. Brain. 98 : 531 - 556.

Hunt, C. C. (1951) The reflex activity of mammalian small-nerve fibres. J. Physiol. 115 : 456 - 469

Rose, D. L. (1975) The decompensated back. Arch. Phys. Med. Rehab. 56 : 51 - 58

Travell, J. (1952) Ethyl chloride spray for painful muscle spasm. Arch. Phys. Med. Rehab. 33 : 291 - 298

Wyke, B. (1967) The neurology of joints. Anns. R.C.S. Eng. 41 : 25 - 50

Wyke, B. (1970) The neurological basis of thoracic spinal pain. Rheum. Phys. Med. 10 : 356 - 367

Wyke, B. (1976) The Lumbar Spine and Back Pain. 189 - 256. Ed. Jayson, M. New York : Grune & Stratton.

5. THE EFFECT OF PASSIVE DORSIFLEXION OF THE FOOT ON THE PASSIVE HAMSTRING STRETCH TEST

INTRODUCTION

The straight leg raising test is part of the routine examination of patients with low back pain and sciatica. A well known modification of this test is first to determine the angle of straight leg raise that causes pain. The leg is then lowered slightly until the pain has gone and the foot is then dorsiflexed. If this reproduces the pain it is generally presumed that the dorsiflexion has increased traction on the sciatic nerve and nerve roots which are probably being irritated by a posterior disc protrusion.

The reliability of the straight raising test has already been questioned (Fisk 1975).

It has been proposed that in some cases of back pain there is evidence of a reflex excitability of the motor neurones in the appropriate segment. This was demonstrated by altered tensions in the hamstring muscles on the painful side, as the straight leg was passively raised. If this is so the gastroc-soleus group of muscles should also be involved. Like the hamstrings they derive their main nerve supply from the fifth lumbar and first sacral segments. This would be useful knowledge because the

gastroc-soleus muscles are more accessable for electrophysiological studies and it would validate their use in further research.

The use of a pulley system incorporating a linear tension gauge, showing how the tension changes as the straight leg is passively raised has been described.

It is important to establish the consistency of these tension gauge measurements, to ensure that repeated stretching of the hamstring muscles, as they occur in these experiments, does not appreciably alter them. It would also be useful information if the amount of stretch taking place in the hamstrings and gastroc-soleus muscles could be approximately calculated as the leg is raised and the foot dorsiflexed.

METHOD

Tension measurements were taken at five degree increments as the leg was passively raised by a pulley system, as previously described. With the first subject (Dr. G. A.), three readings were taken from both legs at half hour intervals. The ankle was left free. Then six consecutive readings were taken as rapidly as possible from the right leg.

At a later date (nineteen days later) the same

subject was used. This time three consecutive readings were taken from the left leg as rapidly as possible. By adjusting the pivoting foot plate of the modified boot, the ankle was then fixed at 85 degrees dorsiflexion and the tension measurements repeated.

Three further subjects were measured with the ankle free and then fixed at varying degrees of dorsiflexion.

Next, a patient with left low back pain was studied both before and after manipulation of the left side of his lumbo-sacral spine. This patient fitted into the previously described clinical pattern. The brief clinical details are as follows:

Mr. J. B.

Aged 45 years. Occupation : police officer.

History of present attack:

Three days before being seen he felt a sudden sharp jab of pain in his left low back whilst lifting a bag out of the boot of his car. This has persisted and there had been no improvement for the past two days.

Past History:

He had noticed that his back had been sore for a number of years after doing work such as digging in the garden, but he had never been off work because of backache.

Examination:

Medium height. Thick set.

Standing:

On forward flexion with the knees straight, in an attempt to touch his toes, palpation of the posterior superior iliac spines showed that the left side did not lift up to the same extent as the right.

Sitting:

When asked to attempt to put his head between his knees the posterior superior spines moved up evenly.

Lying: Supine -

The clinical end point of the passive hamstring stretch was at a lower angle on the left side.

Prone -

There was localised increased muscle tension at the

left lumbosacral level. There was increased tenderness to firm pressure between the fifth lumbar and first sacral posterior spines. There was also a trigger point in his left gluteal muscles.

First, two consecutive readings were taken from both legs with the ankle joint not fixed. Next, one set of readings was taken with the ankle fixed at 90 degrees. Further dorsiflexion was found to be too uncomfortable. The left lumbosacral spine was then manipulated, using the specific technique described elsewhere (see Appendix). The measurements were then repeated after a fifteen minute interval with the ankle free and then fixed at 90 degrees. The patient was remeasured one week later, the measurements being taken with the ankle free. At this time he stated that he was back to normal, only occasionally noticing slight discomfort when he bent forward.

An attempt was made to try and assess the amount of stretch taking place in the hamstring muscles as the leg was being passively raised. Two lateral X-Rays of the same subject were taken from the same position. One was taken with the thigh straight and the other with it flexed to about 40 degrees. These two X-Rays were superimposed. The centre of the head of the femur was taken as the pivot point. From this point it was possible to measure the distance to the tip of the ischial tuberosity

and the angle this made with the femur. The distance from the tip of the greater trochanter to the head of the fibula was measured. From these measurements it was possible to approximately calculate the increase in length that would take place in the hamstring muscles as the leg was being raised.

To measure the change in length taking place in the gastroc-soleus muscles as the foot was dorsiflexed a subject lay prone on the couch and a point on the heel was marked with a ball point pen. Further points 10, 25 and 55 cms. from this point were marked on the back of the leg, the 55cm. mark being above the knee. The foot was dorsiflexed at varying angles, the angles being measured with a spirit level goniometer. The alteration in length between the fixed point on the heel and the other points was measured.

RESULTS

In the first subject the three consecutive readings taken from both legs at half hourly intervals were as shown in Fig. 16.

Fig. 17 shows the six consecutive readings taken from the right leg using the same subject.

Using the same subject at a later date the results of three consecutive readings from the left leg and one set

FIG 17

DR G.A. 6 CONSECUTIVE READINGS TAKEN AS QUICKLY AS POSSIBLE AFTER EACH OTHER (Right leg).

	1	2	3	4	5	6
5°	6.2	6.0	6.0	5.9	6.0	6.0
10°	6.1	5.9	5.9	5.9	5.9	5.9
15°	5.9	5.6	5.7	5.8	5.7	5.8
20°	5.8	5.6	5.6	5.6	5.6	5.6
25°	5.7	5.4	5.5	5.5	5.5	5.5
30°	5.6	5.3	5.4	5.4	5.4	5.4
35°	5.6	5.4	5.4	5.4	5.4	5.3
40°	5.6	5.4	5.4	5.4	5.4	5.3
45°	5.7	5.5	5.5	5.5	5.5	5.5
50°	5.9	5.7	5.6	5.7	5.7	5.6
55°	6.2	6.1	5.9	5.9	5.9	5.9
60°	6.5	6.5	6.3	6.4	6.5	6.3
65°	7.2	7.1	7.1	7.0	7.4	7.1
70°						
	2 mins.	1 min.	1½ mins.	1 min.	1 min.	

TIME INTERVAL BETWEEN THE TAKING OF THE LAST READING.

MEASUREMENTS TAKEN IN KILOGRAMS.

FIG 18

3 CONSECUTIVE READINGS - LEFT LEG

	5°	10°	15°	20°	25°	30°	35°	40°	45°	50°	55°	60°	65°	70°
1	6.2	6.2	6.0	5.8	5.7	5.6	5.6	5.6	5.7	5.9	6.3	7.0		
2	5.9	5.7	5.6	5.5	5.4	5.4	5.4	5.4	5.6	5.7	6.2	6.9		
3	6/1	6.0	5.9	5.8	5.6	5.5	5.4	5.4	5.5	5.9	6.3	6.9		

ANKLE FIXED 85°

6.6	6.4	6.3	6.1	6.0	6.0	6.1	6.2	6.5	6.7				

MEASURED IN KILOGRAMS

FIG 19

	1. DR. R.H. AGED 33 L. LEG			2. MR. P.W. AGED 47 L. LEG		3. MRS. N.D. AGED 25 L. LEG			
	ANKLE FREE	FIXED 85°	FIXED 80°	ANKLE FREE	FIXED 85°	ANKLE FREE	FIXED 90°	FIXED 85°	RECHECK FREE
5°									
10°			6.4				5.8	5.8	
15°			6.0				5.6	5.9	
20°			6.2		6.8		5.8	6.5	
25°	5.4	6.6	7.5		6.8	5.2	6.1	6.9	5.3
30°	5.4	6.4	8.8		7.8	5.2	6.4		5.4
35°	5.5	6.8			8.5	5.4	7.0		5.5
40°	5.9	712				5.6			5.8
45°	6.0	7.8				6.1			6.0
50°	7.0			7.1		7.1			6.8
55°	8.1			7.1					
60°				8.0					
65°									
70°									

MEASURED IN KILOGRAMS

of readings with the ankle dorsiflexed to 85 degrees were as shown in Fig. 18.

Three other subjects were used taking measurements with the ankle free and then fixed at varying angles of dorsiflexion, the results being as shown in Fig. 19.

In order to simplify this table the measurements taken before the tension started to rise are omitted.

These measurements may be illustrated more clearly in the form of a graph, using the measurements from the third subject (see Fig. 20).

The measurements from the patient with left low back pain treated by manipulation were as shown in Fig. 21, the earlier readings again have been omitted.

Pooling and averaging the difference between the last two measurements, taking the non painful side as zero, shows a swing of the painful side towards the non painful side of 1.2 Kgs. and 1.15 Kgs., comparing the two sets of measurements before and after manipulation, with the ankle free. With the ankle fixed at 90 degrees the swing is 0.6 Kgs., but these figures are taken 5 degrees earlier.

The measurements taken from the X-Ray made it possible to calculate that the stretch in the long head of

FIG. 20

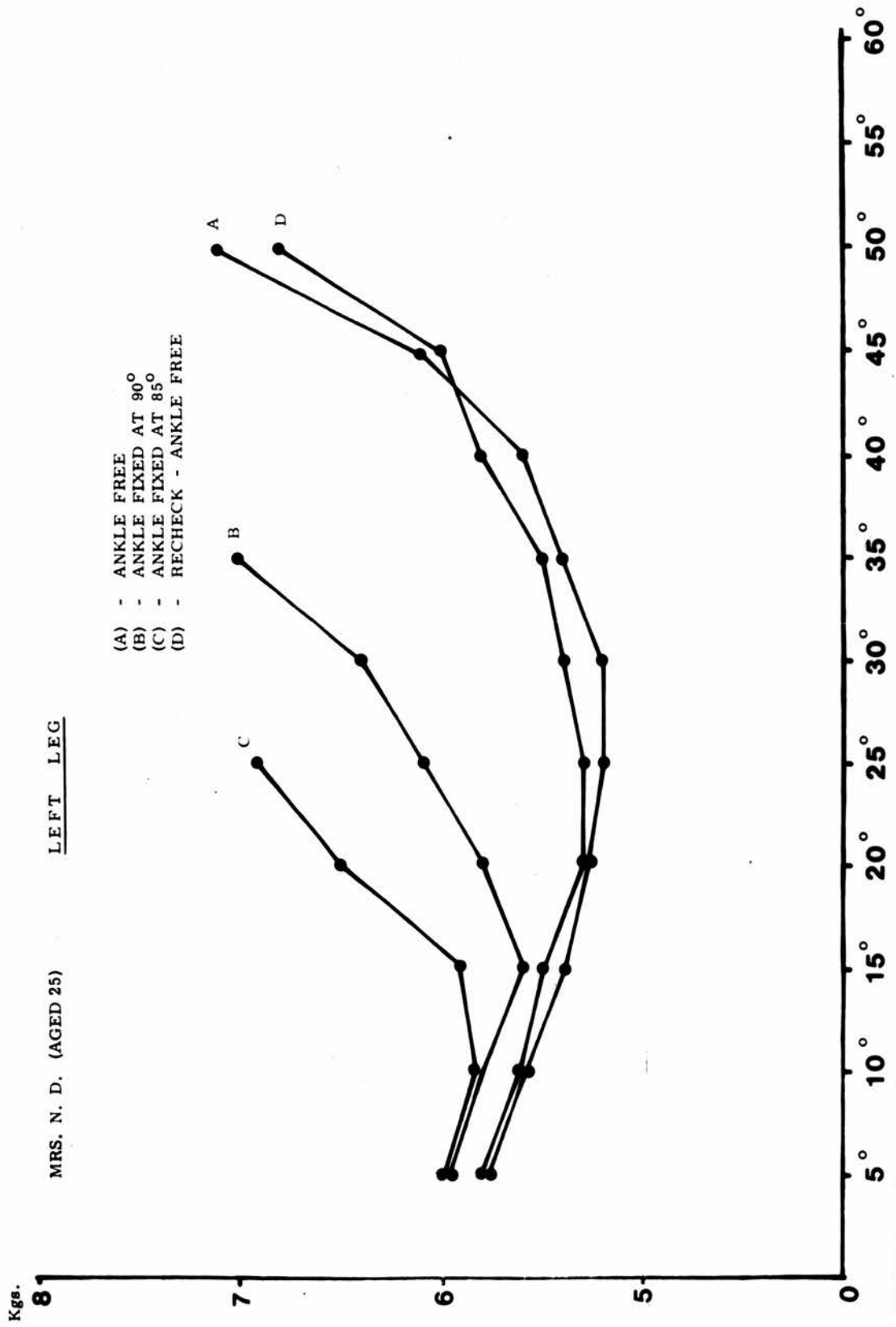
Kgs.
8
7
6
5
0
5°
10°
15°
20°
25°
30°
35°
40°
45°
50°
55°
60°
A
B
C
D
(A) - ANKLE FREE
(B) - ANKLE FIXED AT 90°
(C) - ANKLE FIXED AT 85°
(D) - RECHECK - ANKLE FREE
MRS. N. D. (AGED 25)
LEFT LEG

FIG 21

MR. J.B. LEFT LOW BACK PAIN

	BEFORE MANIPULATION						AFTER MANIPULATION				RECHECK 1 WEEK LATER			
	FIRST TWO MEASUREMENTS ANKLE FREE				ANKLE FIXED 90°		ANKLE FREE		ANKLE FIXED 90°		ANKLE FREE FIRST		ANKLE FREE SECOND	
	L	R	L	R	L	R	L	R	L	R	L	R	L	R
40°	5.6	5.4	4.6	4.9	5.8	5.6	5.1	5.2	5.8	5.6	5.6	5.5	5.6	5.4
45°	5.6	5.4	5.0	4.9	5.8	5.6	5.4	5.3	5.8	5.5	5.6	5.5	5.6	5.4
50°	5.6	5.4	5.0	4.9	6.0	5.8	5.5	5.4	6.9	5.7	5.6	5.5	5.6	5.4
55°	5.8	5.4	5.5	5.2	6.2	5.9	5.4	5.5	6.0	6.0	5.7	5.8	5.8	5.5
60°	6.0	5.9	5.9	5.6	6.9	6.2	6.0	5.8	6.6	6.7	6.0	6.0	6.0	5.8
65°	7.0	6.4	6.6	5.8	7.6	7.2	6.5	6.4	7.3	7.3	6.5	6.2	6.6	6.2
70°	7.8	6.9	7.2	6.6			6.8	7.6			7.0	7.2	7.0	7.2

MEASURED IN KILOGRAMS

the biceps femoris for each 5 degrees increase in leg raise would be approximately one cm. At a rough guess approximately one third of the length of this muscle is tendon. Taking this into account it can be calculated that there is approximately a 3% increase in the resting length of the muscle fibres for each five degree increase in leg raise.

Two consecutive measurements taken from the back of the leg of the same subject as the foot was dorsiflexed were as shown in Fig. 22.

DISCUSSION

The repeated measurements from the same subject show that stretching of the hamstrings at the rate involved in this study does not lead to any appreciable change in the hamstring tension. There are, of course, day to day fluctuations in these tension measurements, depending on the subject's activity, or lack of it. This was demonstrated in the first patient studied (see Fig. 9). The patient concerned spent three days sitting in a conference. This led to a marked reduction in the available passive hamstring stretch. Im this study the repeated measurements from the left leg of the same subject at a later date show a considerable change. In the intervening period he had been doing a lot of running and playing a lot of squash.

It is suggested that further studies of this nature could be of considerable vaâue in assessing the effectiveness

FIG 22

ANGLE OF DORSIFLEXION OF FOOT	DISTANCE FROM FIXED POINT ON HEEL — 1st Reading			DISTANCE FROM FIXED POINT ON HEEL — 2nd Reading		
	10 cms.	25 cms.	55 cms.	10 cms.	25 cms.	55 cms.
100°	11	26.1	56	10.8	26	55.6
95°	11.3	26.6	56.6	11.1	26.3	56.1
90°	11.6	26.9	56.9	11.6	26.6	56.5
85°	12	27.3	57.2	11.9	26.8	56.9

of hamstring stretch exercises. It has been the candidate's experience that a significant number of patients with back problems have very tight hamstring muscles and that a programme of hamstring stretch exercises leads to a reduction of the back pain. A trial study on two such patients showed that after a month of doing these exercises there was a significant alteration in these measurements and a marked improvement in the patients' symptoms. A more objective trial is being planned. It is also suggested that these measurements could be utilised to test the effectiveness of warming-up exercises ddsigned to prevent the all too common hamstring injuries in certain sports.

Figures 18 and 19 demonstrate that fixing the foot at varying angles of dorsiflexion markedly reduces the available passive stretch in the hamstrings. With increasing dorsiflexion it was found difficult to maintain the position of the foot in the modified boot as the leg was raised. The heel tended to slide backwards. This would tend to lessen the effect on the tension measurements. It was also observed that when the foot was not fixed there was a definite increased plantar flexion as the leg was raised. Accurate measurements have not been taken but at 60 degrees of elevation of the leg there was approximately 10 degrees of increased plantar flexion. Even fixing the ankle at 90 degrees led to a very marked alteration in the tension curve (see Fig. 20).

None of these fixed angles of dorsiflexion caused any discomfort to the subjects or the patient. If attempts at increasing the dorsiflexion caused any discomfort a withdrawal reflex was provoked with consequent quadriceps activity and knee flexion. This immediately reduced the tension measurements.

The question to be asked is why does dorsiflexion of the ankle reduce the available stretch in the hamstrings to such an extent? The gastrocnemiae and hamstrings are boty two-joint muscles and are physiological extensor, or antigravity muscles. They both cross the knee joint and are thus potential knee flexors. They must therefore also be involved in the flexor or withdrawal reflex. They also have a common segmental nerve supply.

It is a possibility that the stretch on the gastrocnemiae tendons could lead to stimulation of the Type 1 mechanoreceptors in the posterior joint capsule of the knee (and also the ankle joint), previously described by Wyke (1972), and thus reflexly stimulate the gamma motor nerves to both the gastrocnemiae and hamstrings, but it would seem more likely that the stretching leads to a segmental increase in the motor neurone excitability via increased stimulation from stretching of the muscle spindle receptors.

The measurements taken from the patient with one-

sided low back pain show an equivalent change to that seen in the subjects when the ankle is fixed at 90 degrees (see Fig. 21). The repeated measurements confirm the consistency of the difference between the painful and non painful side. This difference is also evident when the ankle is fixed at 90 degrees. Furthermore the change in tension that takes place after manipulation of the painful side is equally evident in the measurements taken with the ankle free and fixed at 90 degrees. Similar to the trial patients, he demonstrated evidence of increased motor neurone excitability in the form of a trigger point in the gluteal muscles and increased focal tension in the paravertebral muscle at the lumbosacral level. If the paravertebral muscles at the lumbosacral level, the gluteals, and hamstrings, all show evidence of increased muscle excitability then why not also the gastrocnemiae? They all share in common their main nerve supply from the fifth lumbar and first sacral segments.

This would explain why painful restriction of straight leg raising is not always indicative of disc pathology and how raising the leg and dorsiflexing the ankle could be capable of provoking painful muscle spasm.

It would be very satisfying to be able to demonstrate that the percentage increase in the stretch of the gastrocnemiae leads to a corresponding percentage decrease in the hamstring stretch but the available data is insufficient

to come to such a conclusion (see Fig. 22).

Further more elegant neurophysiological research is obviously necessary. The home-made boot and linear tension gauge have just about outlived their usefulness.

REFERENCES

Fisk, J. W. (1975) The straight leg raising test : its relevance to possible disc pathology. N.Z.M.J. 81 : 557 - 560

Fisk, J. W. (1977) A Practical Guide to Management of the Painful Neck and Back. 94 - 101 Springfield : Thomas

Wyke, B. (1972) Articular neurology - a review. Physiotherapy. 58 : 94 - 99

CONCLUSIONS

It is proposed that the passive hamstring stretch test is a useful clinical measurement in the management of patients with spinal pain, and that it should be part of the routine examination. It is demonstrated that tension gauge measurements taken as the straight leg is passively raised is a simple and valid research tool, and that a marked increase in the tension measurements reflects the limit of available passive stretch in the hamstring muscles.

Statistically valid evidence is presented showing that these tension measurements may be different on the two sides of the same patient suffering from one sided low back discomfort, and that in specific cases this difference can be modified by a particular manipulative procedure. This alteration reflects improvement in the patients' symptoms.

The constant presence of trigger points in the gluteal muscles of these patients, the presence of focal increased paravertebral muscle tension, localised spinal tenderness, and the effect of passive dorsiflexion of the foot on the tension gauge measurements provide evidence of a segmental increase in motor neurone excitability. It is proposed that this segmental reflex excitability is mediated through an increase in gamma motor nerve activity (gamma 'gain') and is a likely reason for creating a

situation whereby a painful syndrome could become self perpetuating.

It would seem most likely that manipulation and trigger point treatment have their effect by reducing this gamma 'gain'. It is suggested that manipulation if and when it is effective works through a reflex mechanism that utilises large fibred proprioceptors that are capable of inhibiting gamma motor nerve activity. There are advocates of many different methods of manipulation. It would seem likely that they all have in common this ability to turn down the gamma 'gain', not replacing fragments of disc, unblocking joints, reducing subluxations, or freeing trapped this or that.

Further avenues of research:

The equipment used in these experiments could be more sophisticated, using instantaneous angle and tension recordings as the legs were raised at a predetermined fixed rate, but it is doubtful if this would yield any more useful information.

The basic problem is to measure a difference in the motor neurone excitability on the two sides. This research indicates that the gastroc-soleus group of muscles may be used for comparison. These are accessible for electrical reflex studies (Hoffman or H reflex). The

candidate spent three weeks in February 1977 working with Dr. A. A. Buerger, Ph.D., Assistant Professor of Physical Medicine and Rehabilitation, University of California, Irvine. This was at the invitation of the University, and the time was spent assisting in preliminary H reflex studies. The complete results of these studies are not yet at hand but it is doubtful if they will produced any useful positive information. Due to the many influences, mainly supraspinal, that effect the H reflex, which is notoriously unpredictable, it would be necessary to record results from the two sides simultaneously. This, at the moment, is technically impossible. Furthermore, if the alteration in excitability is mediated through the gamma loop it may be necessary to put the hip extensor muscles on the stretch before the altered excitability may become evident. This would be uncomfortable for both subjects and patients if maintained for any length of time. Perhaps the positioning used by Stevens et al (1974) would be more suitable.

If the reflex excitability is mediated through increased gamma activity this should be demonstrable by altered responses to tendon taps, which should demonstrate the difference between spindle sensitivity on the two sides. Possibly the technique described by Clarke et al (1973) could be adapted. They measure both the force response and E[redacted]M.G. activity in the gastrocnemiae to a measured

achilles tendon tap. Again there would be the technical difficulties involved in obtaining simultaneous results from both sides.

Until it is technically possible to measure directly gamma nerve activity within peripheral nerves it is felt by the candidate that the research most likely to produce worthwhile results would involve simultaneous E.M.G. recordings from both hamstring muscles, which could be quantified, as the patients and controls were flexing forward with the knees straight. This idea is based on the clinical observation that the pelvis on the affected side is held back, presumably by increased activity of the hamstrings on this side.

REFERENCES

Clarke, A. M., Michie, P. T., Glue, L. C. T. (1973) Human phasic reflex response to parameters of a mechanical stimulus as an index of muscle-spindle sensitivity. Medical & Biological Engineering. II : 597 - 602.

Stevens, A., Stijns, H., Rosselle, N., Stappaerts, K., Michels, A. (1974). Slowly stretching the hamstrings and compliance. Preliminary report. Electromyog. Clin. Neurophysiol. 14 : 495 - 496.

APPENDIX

Technique of manipulation used in the trial

Step 1.

The patient lies on her side facing you, near enough to the side of the couch for your own comfort but not so near that she feels she might fall off. The lower knee is flexed to about 45 degrees. One hand flexes the upper leg while the index finger of the other hand palpates the gap between the posterior spine of the fifth lumbar vertebra and the sacrum (see Fig. A). Usually this joint starts to flex when the hip is at right angles, but, in the presence of tight hip extensors, it flexes earlier. It is important that the joint to be gapped should have the two surfaces in full apposition. Hence, it is important to know when flexion of the joint starts to take place. As soon as movement is felt at the lumbosacral joint, the hip is deflexed a fraction, and the toes of the upper leg are tucked in behind the knee of the lower leg (see Fig. B). The joint that is going to be gapped is the one on the upper side of the patient (see Fig. C).

Step 2.

The patient is asked to turn her head and look up to the ceiling. The wrist and elbow of the lower arm are now grasped and pulled towards you. This rotates the patient's trunk. The pull is slightly cephalid. This

puts some extension on the spine. Care must be taken not to overdo this extension, or the lumbar spine gets "locked". While the patient is rotated an eye is kept on the upper knee. As soon as it starts to move upwards, you know that the locking position has been reached.

Step 3.

The hand nearest the patient's head is placed across her back, so that the thumb presses down on the fifth lumbar spinous process. The elbow lies in the patient's axilla. Care has to be taken not to press hard on the shoulder joint. It helps if the patient's upper arm is pulled forward, making contact with your upper arm (see Fig. D). This arm acts as the long lever. This position may be impossible, if the patient has a long back and you have short arms. The thumb on the spinous process is not necessary and adds little to the counter thrust. Its main function is to help in sensing which joint has been gapped. If it is impossible to reach this level, placing the palm of the hand and fingers along the spine will do.

Step 4.

Leaning over the patient, the other forearm is placed across her buttock so that it lies on the groove between the hip joint and the pelvic crest. This forearm provides the short-amplitude, high velocity thrust. It is very important to be comfortably balanced over the patient,

so that this thrust can be applied. (see Fig. E.)

Step 5.

The knee nearest the patient's head rests against the side of the couch. The other knee is placed over the patient's upper flexed knee (see Fig. F). Downward pressure with this knee rotates the patient's pelvis towards you and increases the rotation of the spine. This augments the thrust from the short lever.

Step 6.

Pressure is gradually increased on the patient's knee and pelvis until it is felt that you have taken up all the slack. The upper trunk is prevented from further rotation by the long lever. It is helpful to gently and smoothly rock the patient, using the three points of contact to control the rocking, and while rocking to gradually increase the counterpressure between the two levers. The direction of pressure and counterpressure from the two levers is more effective with a downwards direction in the lower short lever and an upwards direction in the long lever. This is in addition to pressure at right angles to the trunk, and will provide a force more at right angles to the lumbosacral joint.

Step 7.

When it is felt that all the slack has been taken up, instruct the patient to take a deep breath, blow it all out, and to go limp. The extra slack is taken up by the two levers. At the height of exhalation, a short, high-velocity thrust is applied with the short lever. This should pop the joint on the patient's upper side.

(This is taken almost word for word from Fisk (1977) 'A Practical Guide to Management of the Painful Neck and Back', pages 94 - 99. Reproduced by kind permission of the Publisher, Charles C. Thomas : Illinois.)

FIG. A

FIG. B

FIG. C

FIG. D

FIG. E

FIG. F

TABLE ⊗ CONTROLS

			30°	35°	40°	45°	50°	55°	60°	65°	70°
(1) MALE 26 YRS	BEFORE MANIPULATION	LEFT	5.5	5.4	5.5	5.6	5.8	6.0	6.5		
		RIGHT	5.4	5.5	5.6	5.7	5.8	6.2	6.7		
	AFTER RIGHT MANIPULATION	LEFT	5.6	5.5	5.5	5.6	5.8	6.1	6.6	4½ MIN. AFTER	
		RIGHT	5.5	5.5	5.5	5.5	5.5	6.0	6.4	6½ MIN. AFTER	
	AFTER LEFT MANIPULATION	LEFT	5.4	5.3	5.4	5.5	5.6	6.0	6.4	6 MIN. AFTER	
		RIGHT	5.5	5.3	5.4	5.5	5.8	6.1	6.7	4 MIN. AFTER	
(2) MALE 26 YRS	BEFORE MANIPULATION	LEFT	6.7	6.6	6.5	6.5	6.7	7.0	7.5		
		RIGHT	6.8	6.7	6.6	6.7	6.7	6.9	7.1		
	AFTER LEFT MANIPULATION	LEFT	6.6	6.5	6.5	6.4	6.6	6.7	7.1	6 MIN. AFTER	
		RIGHT	6.5	6.5	6.5	6.5	6.5	6.6	7.0	3½ MIN. AFTER	
	AFTER RIGHT MANIPULATION	LEFT	6.6	6.6	6.5	6.6	6.6	6.9	7.4	4 MIN. AFTER	
		RIGHT	6.4	6.3	6.3	6.3	6.5	6.7	7.1	6½ MIN. AFTER	
(3) MALE 24 YRS	BEFORE MANIPULATION	LEFT	5.5	5.3	5.2	5.3	5.4	5.6	5.9		
		RIGHT	5.5	5.5	5.4	5.4	5.4	5.5	5.7		
	AFTER LEFT MANIPULATION	LEFT	5.5	5.4	5.3	5.3	5.4	5.6	5.9	6 MIN. AFTER	
		RIGHT	5.7	5.5	5.4	5.4	5.5	5.5	5.8	4 MIN. AFTER	
	AFTER RIGHT MANIPULATION	LEFT	5.5	5.5	5.2	5.2	5.2	5.4	5.8	4 MIN. AFTER	
		RIGHT	5.6	5.4	5.3	5.3	5.3	5.4	5.7	5½ MIN. AFTER	
(4) MALE 22 YRS	BEFORE MANIPULATION	LEFT	6.8	7.2	7.6	7.9	8.2				
		RIGHT	7.1	7.3	7.5	8.0	8.4				
	AFTER LEFT MANIPULATION	LEFT	6.7	6.9	7.2	7.6	7.8	7 MINUTES AFTER			
		RIGHT	6.8	6.9	7.1	7.4	7.6	3½ MINUTES AFTER			
	AFTER RIGHT MANIPULATION	LEFT	6.9	7.1	7.3	7.9	8.2	4 MINUTES AFTER			
		RIGHT	7.1	7.4	7.6	7.9	8.4	6½ MINUTES AFTER			
(5) MALE 47 YRS	BEFORE MANIPULATION	LEFT	5.3	5.2	5.1	5.1	5.1	5.3	5.8		
		RIGHT	5.3	5.2	5.1	5.1	5.1	5.4	6.2		
	AFTER LEFT MANIPULATION	LEFT	5.3	5.2	5.1	5.0	5.2	5.3	6.0	7 MIN. AFTER	
		RIGHT	5.3	5.3	5.2	5.2	5.3	5.6	6.4	5 MIN. AFTER	
	AFTER RIGHT MANIPULATION	LEFT	5.4	5.3	5.2	5.2	5.2	5.4	5.7	4½ MIN. AFTER	
		RIGHT	5.3	5.2	5.1	5.1	5.1	5.6	6.2	6 MIN AFTER	
(6) MALE 41 YRS	BEFORE MANIPULATION	LEFT	5.5	5.2	5.2	5.1	5.1	5.2	5.5	6.0	
		RIGHT	5.5	5.4	5.4	5.4	5.3	5.5	5.6	6.2	
	AFTER LEFT MANIPULATION	LEFT	5.3	5.2	5.1	5.1	5.1	5.3	5.5	6.4	6 MIN.AFT.
		RIGHT	5.5	5.4	5.4	5.4	5.6	5.5	5.8	6.1	8½MIN.AFT.
	AFTER RIGHT MANIPULATION	LEFT	5.4	5.3	5.2	5.1	5.3	5.4	5.7	6.4	5½MIN.AFT.
		RIGHT	5.4	5.3	5.3	5.3	5.3	5.5	5.7	6.1	8 MIN.AFT.

(7) MALE 25 YRS	BEFORE MANIPULATION	LEFT	6.0	5.9	6.0	6.1	6.4	6.9				
		RIGHT	6.2	6.1	6.1	6.1	6.5	6.9				
	AFTER LEFT MANIPULATION	LEFT	6.0	6.0	6.0	6.2	6.4	6.7	9½ MINUTES AFTER			
		RIGHT	6.2	6.0	6.0	6.0	6.2	6.6	4½ MINUTES AFTER			
	AFTER RIGHT MANIPULATION	LEFT	6.1	6.0	6.1	6.2	6.4	6.9	4 MINUTES AFTER			
		RIGHT	6.1	6.0	5.9	6.1	6.3	6.7	6 MINUTES AFTER			
(8) MALE 25 YRS	BEFORE MANIPULATION	LEFT	6.0	5.9	5.9	5.9	6.1	6.3	6.7	7.2		
		RIGHT	6.0	5.9	5.9	5.9	5.9	5.9	6.2	6.8		
	AFTER RIGHT MANIPULATION	LEFT	6.0	6.0	6.0	6.0	6.2	6.5	6.9	7.4	5 MIN AFT.	
		RIGHT	5.9	5.9	5.8	5.7	5.7	5.7	5.9	6.1	6.9	7½ MIN AFT.
	AFTER LEFT MANIPULATION	LEFT	6.0	5.9	5.9	5.9	6.1	6.3	6.8	7.5	6½ MIN AFT.	
		RIGHT	6.0	5.9	5.9	5.9	5.9	6.0	6.2	6.7	6 MIN AFT.	
(9) FEMALE 46 YRS	BEFORE MANIPULATION	LEFT	4.7	4.6	4.7	5.0	5.1	5.4	6.0	6.3		
		RIGHT	4.9	4.9	4.8	5.0	5.1	5.3	5.7	6.0		
	AFTER RIGHT MANIPULATION	LEFT	5.0	5.0	5.0	5.1	5.2	5.5	5.8	6.3	4½ MIN AFT.	
		RIGHT	4.8	4.7	4.6	4.8	5.0	5.4	6.0	6.4	6½ MIN AFT.	
	AFTER LEFT MANIPULATION	LEFT	4.9	5.0	5.1	5.2	5.8	6.2	7.0	7.6	6½ MIN AFT.	
		RIGHT	4.9	4.8	4.9	5.0	5.2	5.6	6.0	6.7	6 MIN AFT.	
(10) MALE 30 YRS	BEFORE MANIPULATION	LEFT	5.1	5.0	4.9	4.9	5.0	5.1	5.3	5.9	6.0	
		RIGHT	4.7	4.7	4.6	4.7	4.5	4.7	5.0	5.3	5.9	
	AFTER LEFT MANIPULATION	LEFT	4.9	4.9	4.9	4.8	4.8	4.9	5.1	5.7	6.1	8 MIN AFT.
		RIGHT	5.3	5.3	5.3	5.3	5.3	5.3	5.4	5.9	6.5	4 MIN AFT.
	AFTER RIGHT MANIPULATION	LEFT	5.1	5.0	4.9	4.9	4.9	5.1	5.2	5.7	6.2	5 MIN AFT.
		RIGHT	5.2	5.1	5.1	5.1	4.9	4.9	5.4	5.6	6.0	8 MIN AFT.

To simplify this Table the first five readings have been omitted.

TABLE . PATIENTS

			30°	35°	40°	45°	50°	55°	60°	65°	70°	75°
(1) MALE 31 YRS	BEFORE MANIPULATION	LEFT	5.8	5.8	5.7	5.9	5.8	6.0	7.3			
		RIGHT	5.6	5.5	5.7	6.0	6.9	7.4				
	AFTER LEFT MANIPULATION	LEFT	5.9	6.1	6.5	6.5	6.3	6.8	7.8	8.4		
		RIGHT	5.6	5.7	5.8	6.2	7.2	8.2				
RIGHT PAIN	AFTER RIGHT MANIPULATION	LEFT	5.8	5.8	5.8	6.0	6.2	7.0	7.4	8.6		
		RIGHT	5.3	5.4	5.7	5.8	5.7	5.9	6.3	7.2		
(2) FEMALE 31 YRS	BEFORE MANIPULATION	LEFT	3.6	3.4	3.8	3.8	3.8	4.0	4.5	5.0		
		RIGHT	4.0	3.6	3.8	3.8	3.8	4.0	4.0	4.2	5.0	
	AFTER RIGHT MANIPULATION	LEFT	3.8	3.6	3.6	3.6	3.6	4.0	4.3	5.0	5.8	
		RIGHT	3.5	3.5	3.6	3.6	3.6	3.6	3.8	4.2	4.8	5.5
LEFT PAIN	AFTER LEFT MANIPULATION	LEFT	3.6	3.6	3.4	3.4	3.4	3.6	4.0	4.4	4.8	5.6
		RIGHT	3.4	3.4	3.4	3.4	3.6	3.4	4.0	4.4	5.0	6.0
(3) MALE 40 YRS	BEFORE MANIPULATION	LEFT	5.8	5.5	5.3	5.2	5.2	5.2	5.4	6.0	7.0	
		RIGHT	5.8	5.6	5.6	5.5	5.4	5.4	5.2	5.7	6.0	6.4
	FAILED TO MANIPULATE RIGHT SIDE											
LEFT PAIN	AFTER LEFT MANIPULATION	LEFT	5.7	5.4	5.3	5.0	4.9	5.1	5.2	5.5	5.9	6.4
		RIGHT	5.8	5.4	5.2	5.4	5.4	5.4	5.6	6.9	6.2	6.2
(4) MALE 39 YRS	BEFORE MANIPULATION	LEFT	6.0	5.8	5.5	5.4	5.4	5.6	5.8	5.8	6.4	7.2
		RIGHT	7.5	7.0	6.8	6.8	6.8	6.8	6.8	6.8	7.2	7.8
	AFTER LEFT MANIPULATION	LEFT	5.8	5.8	5.6	5.4	5.4	5.4	5.8	6.0	7.0	7.8
		RIGHT	6.4	6.6	6.5	6.5	6.5	6.5	6.5	6.6	7.1	8.0
RIGHT PAIN	AFTER RIGHT MANIPULATION	LEFT	5.9	5.7	5.6	5.4	5.4	5.7	5.8	6.0	7.0	8.2
		RIGHT	6.1	6.2	6.0	6.2	6.0	6.0	6.2	6.4	7.1	8.1
(5) MALE 39 YRS	BEFORE MANIPULATION	LEFT	7.3	7.3	7.2	6.7	6.9	6.9	7.1	7.6	8.1	
		RIGHT	7.3	7.1	7.1	7.0	6.9	6.9	7.2	7.4	7.9	8.2
	AFTER RIGHT MANIPULATION	LEFT	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.7	8.2	
		RIGHT	7.6	7.4	7.3	7.3	7.4	7.3	7.5	7.1	7.5	8.2
LEFT PAIN	AFTER LEFT MANIPULATION	LEFT	7.2	7.2	7.2	7.2	7.2	7.2	7.2	7.8	7.9	8.6
		RIGHT	7.1	6.9	6.8	6.9	7.0	6.9	6.9	7.5	8.2	
(6) FEMALE 45 YRS	BEFORE MANIPULATION	LEFT	4.6	4.4	4.4	4.5	4.6	4.6	5.0	5.6		
		RIGHT	4.5	4.3	4.3	4.4	4.3	4.3	4.5	4.7	6.2	
	AFTER RIGHT MANIPULATION	LEFT	4.6	4.4	4.4	4.2	4.2	4.2	4.4	4.8	5.2	
		RIGHT	4.5	4.3	4.3	4.2	4.1	4.1	4.3	4.5	5.1	
LEFT PAIN	AFTER LEFT' MANIPULATION	LEFT	4.4	4.3	4.1	4.1	4.1	4.1	4.2	4.6	5.2	
		RIGHT	4.7	4.4	4.3	4.2	4.1	4.1	4.1	4.4	4.9	

(7) MALE 55 YRS RIGHT PAIN	BEFORE MANIPULATION	LEFT	5.6	5.3	5.2	5.1	5.1	5.0	5.3	5.4	5.8	6.6
		RIGHT	5.7	5.4	5.3	5.2	5.4	5.7	5.8	6.2	7.3	
	AFTER DIRECT THRUST (CAPPING) (L) FAILED	LEFT	5.8	5.6	5.3	5.1	5.0	5.0	4.9	5.1	6.5	6.3
		RIGHT	5.8	5.6	5.4	5.3	5.2	5.1	5.1	5.1	5.2	
	AFTER RIGHT MANIPULATION	LEFT	5.8	5.5	5.3	5.2	5.1	5.1	5.3	5.6	6.0	6.7
		RIGHT	5.6	5.4	5.2	5.2	5.0	5.0	5.0	5.1	5.3	
(8) MALE 25 YRS RIGHT PAIN	BEFORE MANIPULATION	LEFT	6.2	6.1	6.1	6.1	6.2	6.4	7.1			
		RIGHT	6.5	6.5	6.6	6.8	6.8	6.8	7.5			
	AFTER LEFT MANIPULATION	LEFT	6.5	6.5	6.4	6.3	6.5	6.9	7.4			
		RIGHT	7.0	7.0	7.0	7.2	7.3	7.9	8.4			
	AFTER RIGHT MANIPULATION	LEFT	6.6	6.6	6.6	6.6	6.9	7.2	8.1			
		RIGHT	6.4	6.4	6.4	6.6	6.6	6.7	7.4			
(9) FEMALE 37 YRS LEFT PAIN	BEFORE MANIPULATION	LEFT	4.1	4.1	4.1	4.1	4.0	4.1	4.1	4.5	4.9	6.0
		RIGHT	4.2	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.2	4.8
	AFTER RIGHT MANIPULATION	LEFT	4.3	4.2	4.1	4.0	4.0	4.1	4.2	4.6	5.0	5.6
		RIGHT	4.1	4.1	4.1	4.0	4.0	4.0	3.9	4.0	4.3	4.9
	AFTER LEFT MANIPULATION	LEFT	4.0	4.0	4.0	4.0	3.9	3.9	4.1	4.3	4.9	5.8
		RIGHT	4.1	4.1	4.0	4.0	4.0	4.0	4.1	4.3	4.7	5.4
(10) MALE 29 YRS LEFT PAIN	BEFORE MANIPULATION	LEFT	6.9	6.8	6.8	6.8	7.0	7.6	8.2			
		RIGHT	6.8	6.7	6.5	6.6	6.9	7.0	7.6	7.9		
	AFTER RIGHT MANIPULATION	LEFT	6.8	6.7	6.7	6.8	6.9	7.3	8.1			
		RIGHT	6.9	6.8	6.7	6.7	6.7	7.4	7.3	7.9		
	AFTER LEFT MANIPULATION	LEFT	7.1	7.1	7.1	7.2	7.4	7.8	8.3			
		RIGHT	7.0	6.9	7.0	6.9	7.1	7.7	8.5			

To simplify this Table the first five readings have been omitted.